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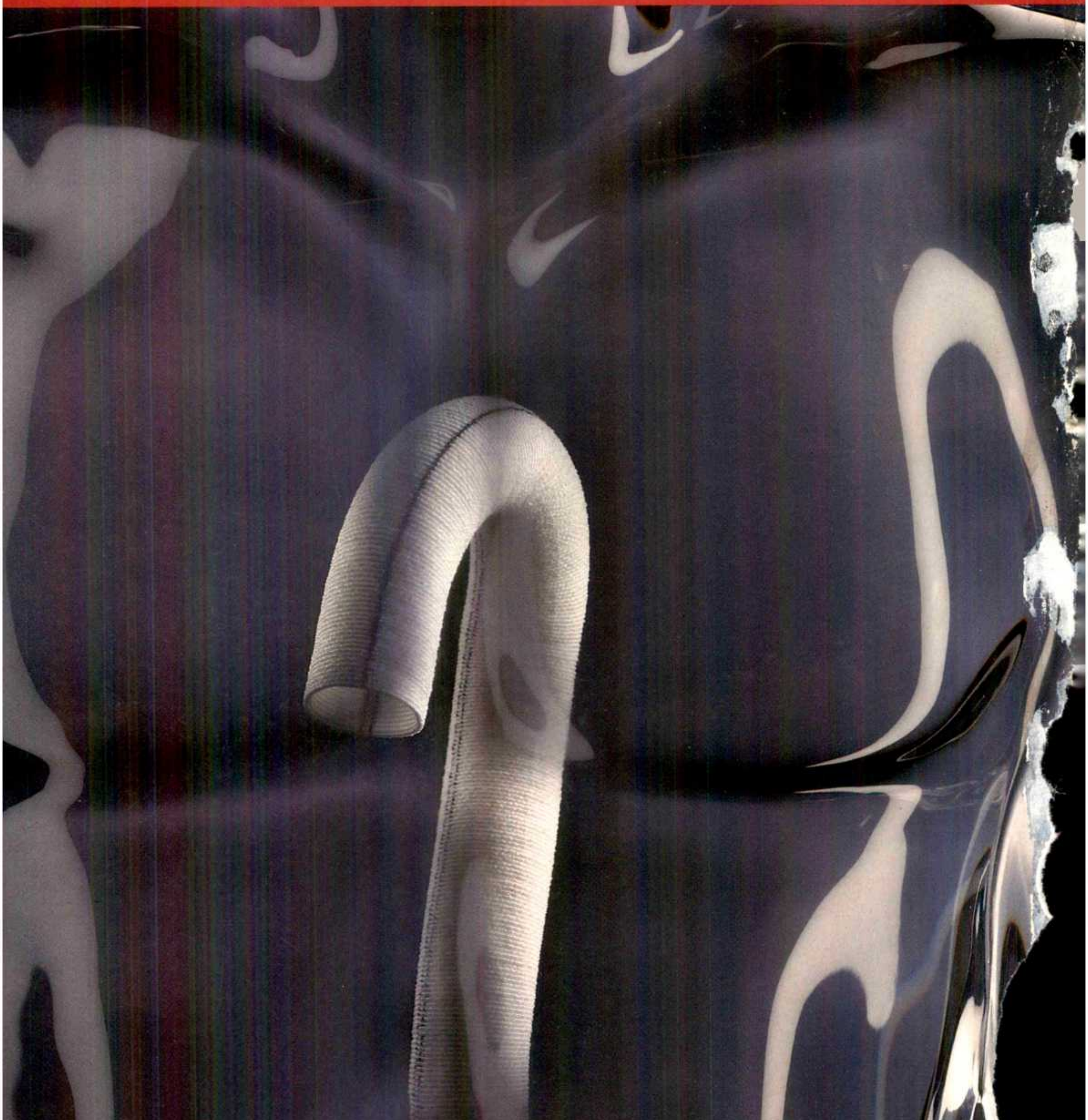

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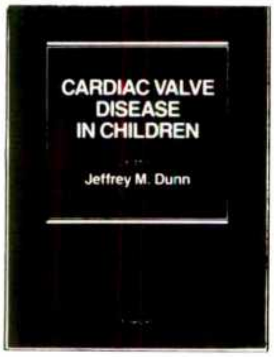

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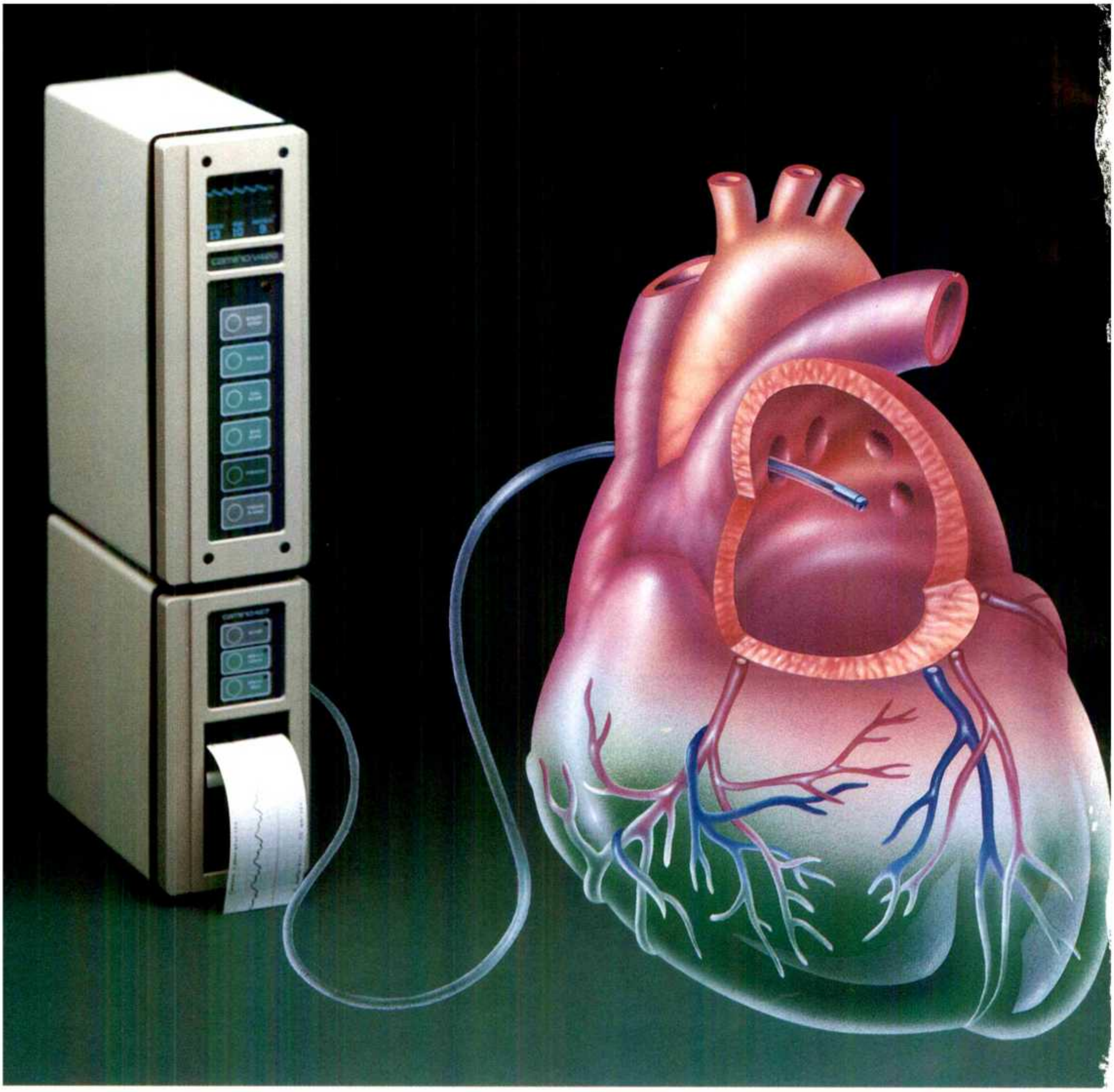

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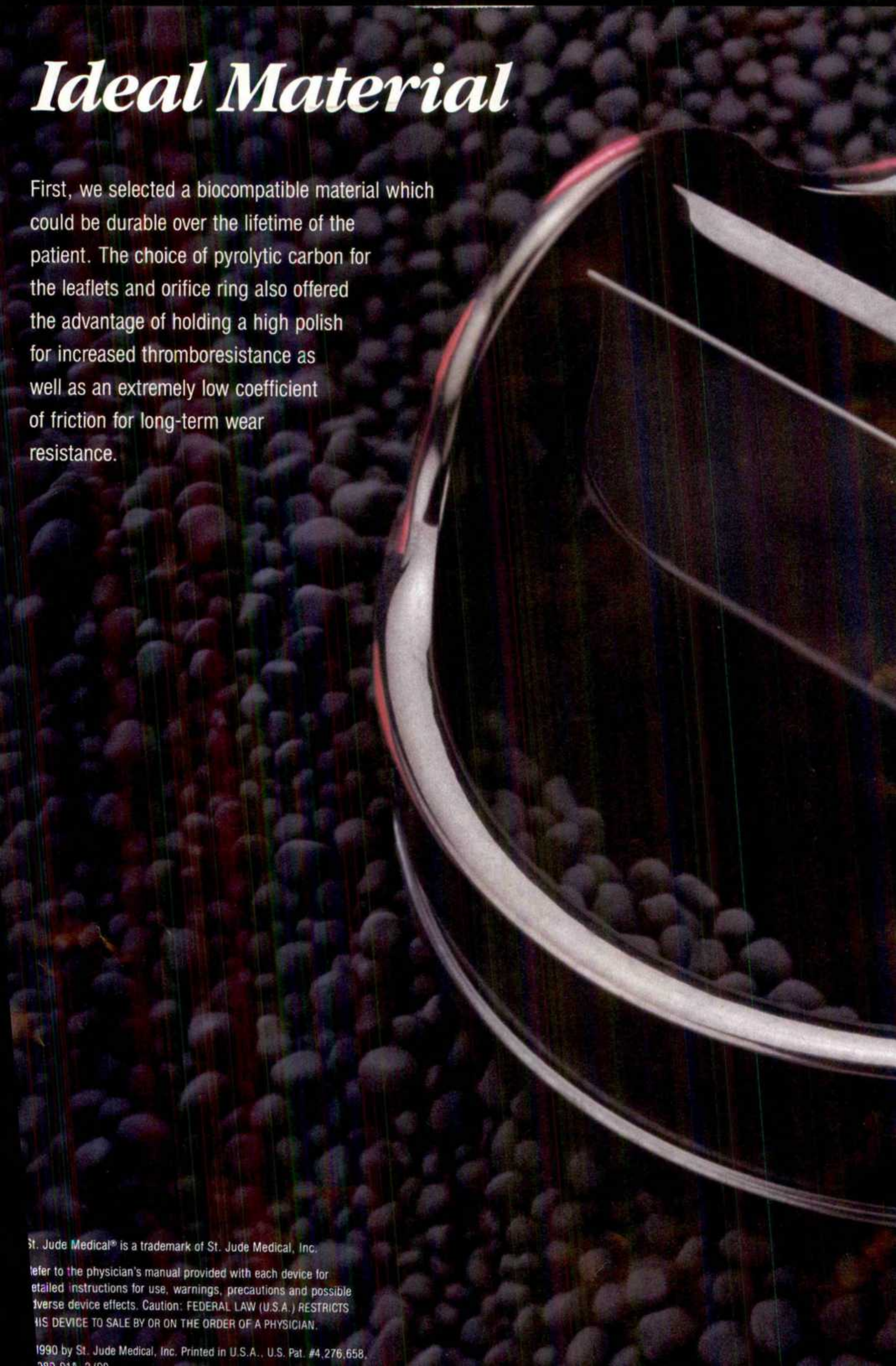

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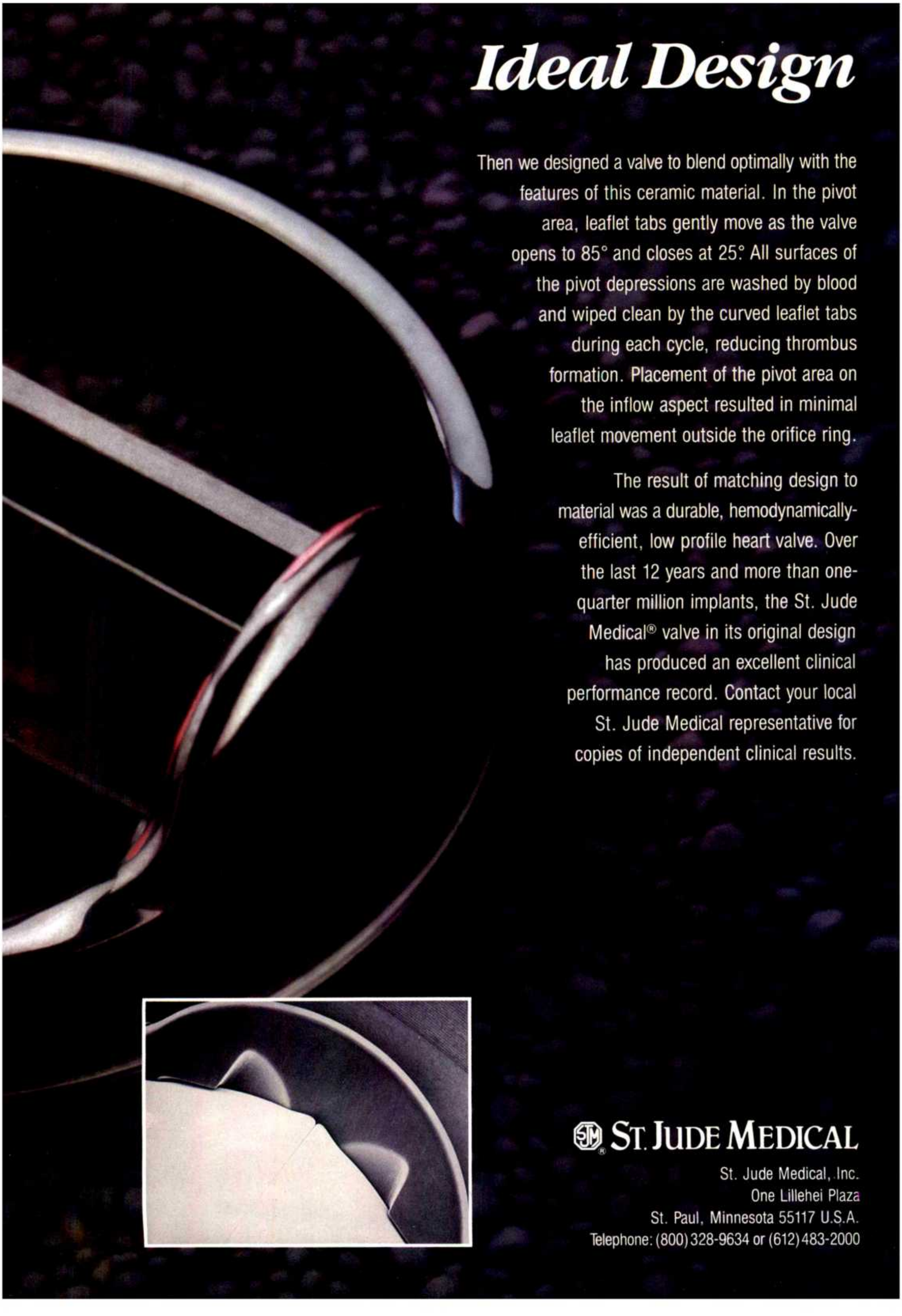

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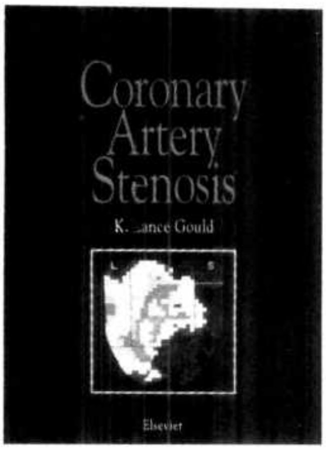

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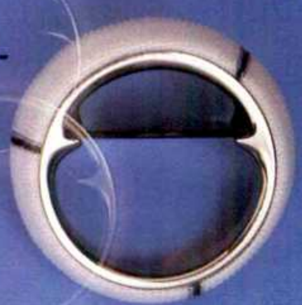

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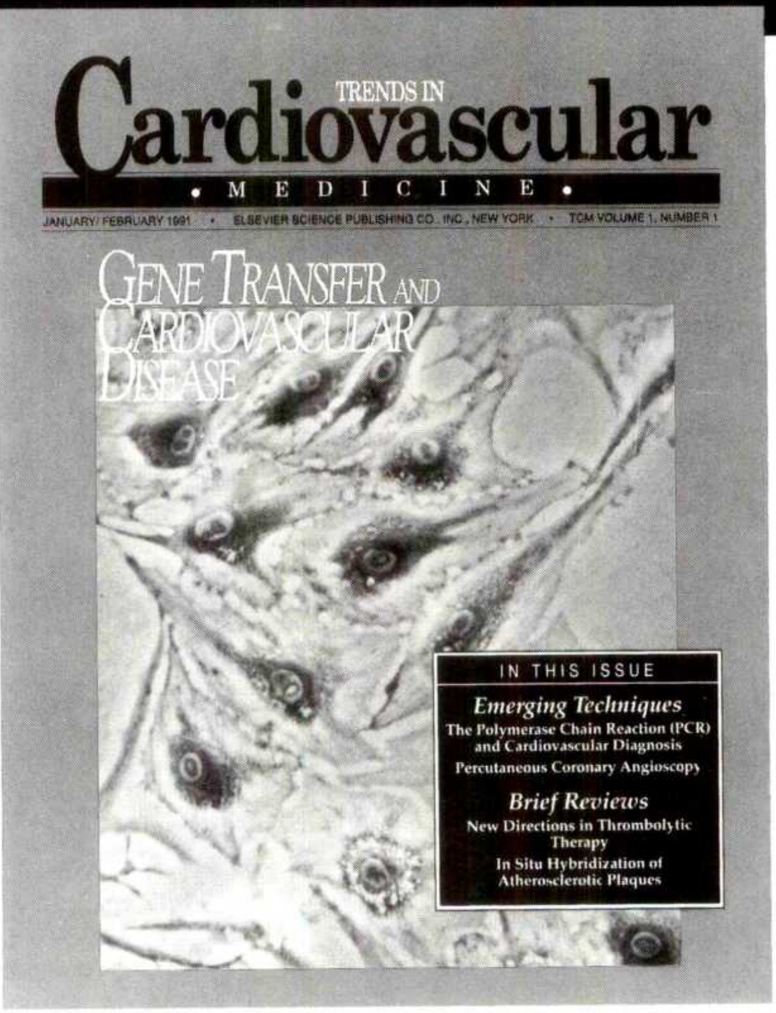

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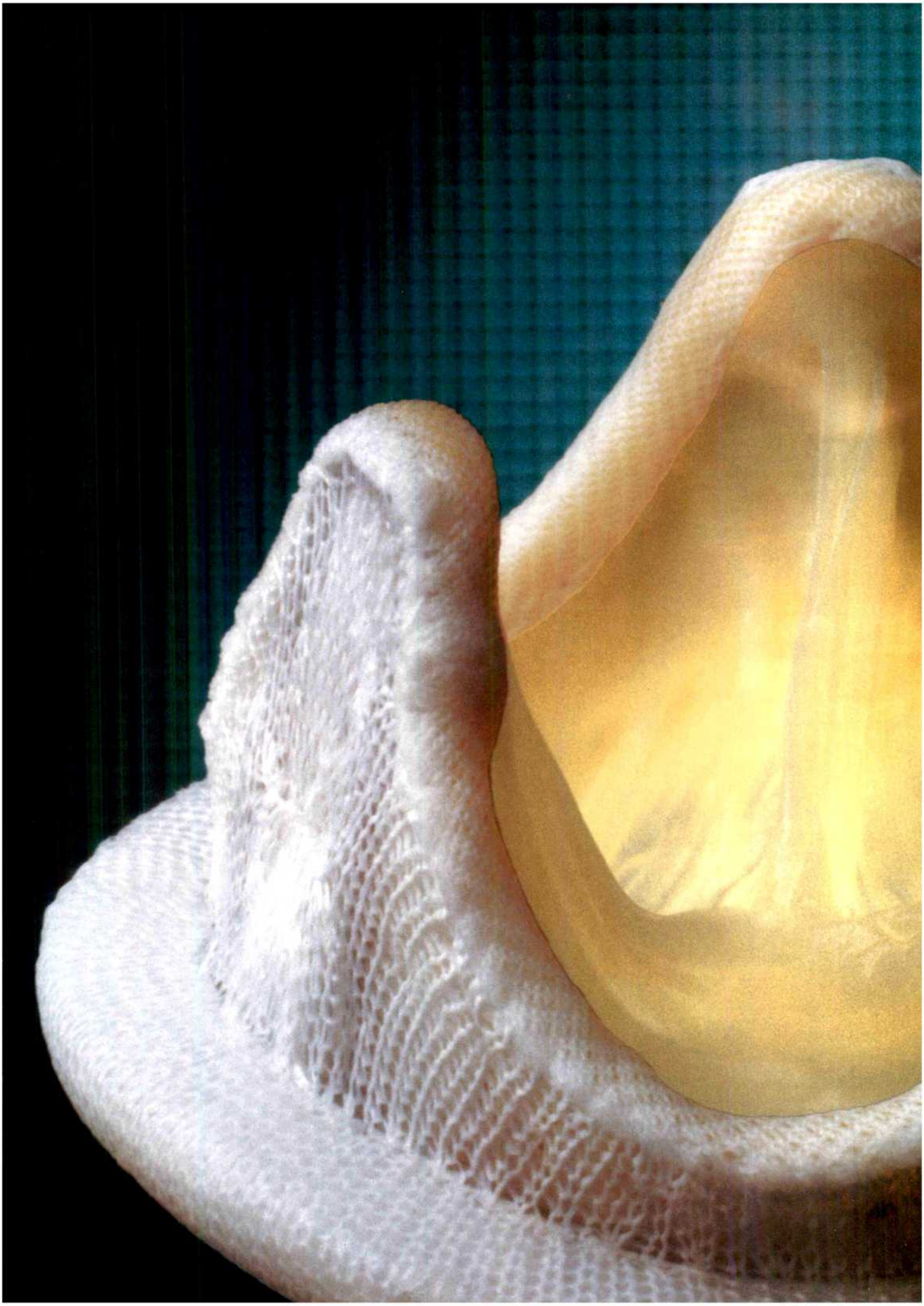

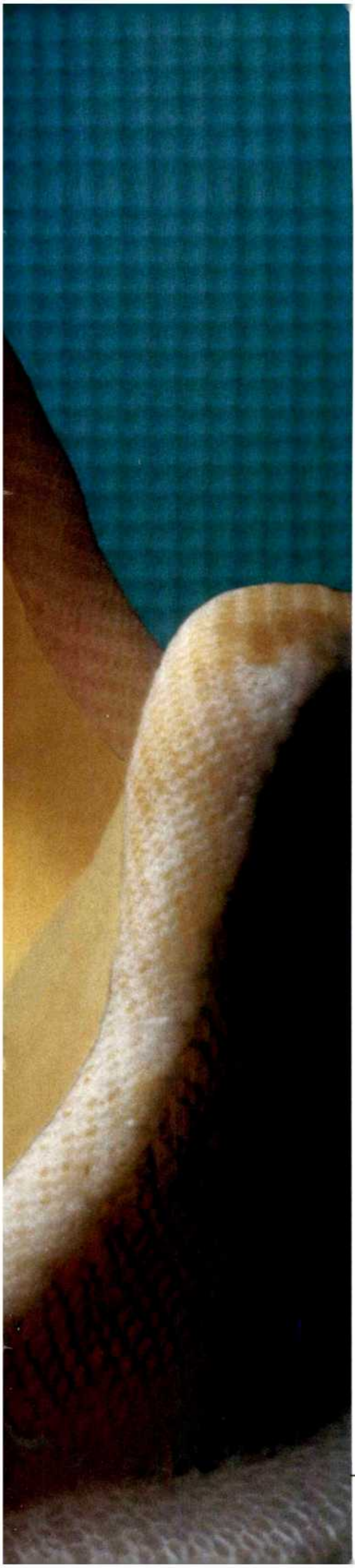

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The decision to use a tissue valve must ultimately be made by the physician on an individual basis after a careful evaluation of the short- and long-term risks and benefits to the patient are made, and alternative methods of treatment are considered. Overall durability, especially long-term, has not been established for bioprostheses. Serious complications, sometimes leading to replacement of the valve and/or death, may be associated with the use of prosthetic valves (see Complications). A full explanation of the benefits and risks should be given to each prospective patient before surgery. **Note:** Bioprostheses should be used with caution in the presence of severe systemic hypertension or when the anticipated patient longevity is longer than the known longevity of the prosthesis. Careful and continuous medical follow-up (at least by an annual visit to the physician) is advised so that valve-related complications, particularly those related to material failure, can be diagnosed and properly managed. Recipients of prosthetic heart valves who are undergoing dental procedures should receive prophylactic antibiotic therapy to minimize the possibility of prosthetic infection. Bioprosthetic heart valve recipients should be maintained on anticoagulant therapy, except where contraindicated, during the initial healing stages after implantation, approximately 2 to 3 months. Anticoagulants should then be discontinued over a period of 10 days, except in those patients for whom indefinite anticoagulant protection is indicated, i.e., in the absence of sinus rhythm and in patients with a dilated left atrium, calcification of the atrial wall or history of previous atrial thrombosis. The appropriate anticoagulation therapy must be determined by the physician on an individual basis. **Adequate rinsing with physiological saline, as described in the Technique section, is mandatory before implantation to reduce the glutaraldehyde concentration. No other solutions, drugs, chemicals, antibiotics, etc. should ever be added to the glutaraldehyde or rinse solutions, as irreparable damage to the leaflet tissue, which may not be apparent under visual inspection, may result. The Carpentier-Edwards bioprostheses must be kept moist at all times. Drying out will cause irreversible damage to the leaflet tissue. To prevent drying out during implantation, the valves should be irrigated periodically on both sides with sterile physiological saline.** Passing diagnostic catheters and transvenous pacing leads across a Carpentier-Edwards bioprosthesis could damage the valve. Consequently, it is recommended that these procedures be avoided. Both the valve and glutaraldehyde storage solution are **sterile.** The outside of the jar is **not sterile** and must **not** be placed in the sterile field. Furthermore, the valve and container should **never** be subjected to sterilization procedures involving ethylene oxide, propylene oxide, steam, or irradiation. Storage between 10ºC and 25ºC is recommended; however, care should be exercised to avoid freezing or extreme heat, which may damage the valvular tissue. **Any valve that has been frozen or exposed to temperatures greater than 60ºC should not be used for human implantation.** Glutaraldehyde may cause irritation of the skin, eyes, nose, and throat, and may also cause skin sensitization. Avoid prolonged or repeated contact or prolonged breathing of the vapor. Use only with adequate ventilation. In the event of contact, immediately flush the affected area with water. In the event of contact with eyes, seek medical attention. **Surgical Precautions: 1.** When choosing a valve for a given patient, the size, age, and physical condition of the patient in relation to the size of the prosthesis must be taken into consideration to minimize the possibility of obtaining a suboptimal hemodynamic result. The selection of a valve, however, must ultimately be made by the physician on an individual basis after carefully weighing all of the risks and benefits to the patient. **2.** Due to the relative flexibility of the frame, care must be exercised to avoid the use of too large a prosthesis to prevent folding or extreme deformation of the stent that may render the valve incompetent. Oversizing may create highly localized mechanical stresses resulting in tissue failure in the form of detachment of the leaflets or porcine aortic wall from the valve stent posts. **3.** Left ventricular perforation or ulceration has been reported. Both open and closed chest cardiac massage in patients with an open strut mitral prosthesis increases the risk of ventricular perforation. Additionally, lifting, flexing, and manual compression of the distended heart after discontinuation of bypass may cause a commissure strut to be pushed through the endocardium, producing a dissecting hematoma with subsequent uncontrolled hemorrhage. **4.** The spacing of the sutures in the remnant of the valvular orifice and the prosthesis suture ring must be carefully matched to avoid folding of the leaflets or extreme distortion of the orifice. Edwards CVS Division has received reports in which individual mattress sutures, spanning a distance of 10 to 15mm, produced a purse-string effect causing compression of the valve orifice. **5.** When using interrupted sutures, it is important to cut the sutures close to the knots and to ensure that exposed suture tails will not come into contact with the leaflet tissue. Cases have been reported in which bioprostheses developed severe regurgitation and had to be replaced as a result of wear due to contact with sutures. **6.** Unlike rigid mechanical valves, the stent wall is soft and will not resist needle penetration. Accordingly, extreme care must be exercised when placing sutures through the sewing margin to avoid penetration of the side wall of the stent and possible laceration of the leaflet tissue. **7.** As with all prostheses that have open cages, free struts, or commissure supports, care must be exercised, particularly in mitral replacement, to avoid looping or catching a suture around the free legs which would interfere with proper valvular function. **8.** The stent of the bioprosthesis is asymmetrical to conform to the natural shape of the porcine aortic valve. Consequently, the commissure supports are not equidistantly spaced. The contrasting suture markers in the sewing ring of the mitral valve denote the greatest intercommissural distance. These two commissure supports should be oriented so as to straddle the ventricular outflow tract to avoid murmurs due to blood flow turbulence. **Note:** The contrasting suture material should not be removed from the prosthesis. **9.** In the aortic position, the struts should correspond to the remnants of the natural commissures so as not to obstruct the coronary ostia. **10.** A serial number tag is attached to the sewing ring of each valve by a suture. This serial number should be checked against the number on the jar; if any difference is noted, the valve should be returned unused. This tag should not be detached from the valve until implant is imminent. Care should be exercised to avoid cutting or tearing the suture ring cloth during removal. **11.** Gentle handling is required for all implantable devices. If the valve is dropped, damaged, or mishandled in any way, it must not be used for human implantation. **Complications:** As with all prosthetic heart valves, serious complications, sometimes leading to death, may be associated with the use of tissue valves. In addition, complications due to individual patient reaction to an implanted device, or to physical or chemical changes in the components, particularly those of biological origin, may occur at varying intervals (hours or days) necessitating reoperation and replacement of the prosthetic device. Complications associated with the use of Carpentier-Edwards bioprostheses include stenosis, regurgitation through an incompetent valve, perivalvular leak, endocarditis, hemolysis, thromboembolism, thrombotic obstruction, bleeding diatheses related to the use of anticoagulant therapy, ventricular perforation by stent posts, looped sutures, and malfunctions of the valve due to distortion at implant or physical or chemical deterioration of valve components. Types of tissue deterioration include infection, calcification, thickening, perforation, suture abrasion, instrument trauma, and leaflet or aortic wall detachment from the valve stent posts. These complications may present clinically as abnormal heart murmur, shortness of breath, exercise intolerance, dyspnea, orthopnea, anemia, fever, arrythmia, hemorrhage, transient ischemic attack, stroke, paralysis, low cardiac output, pulmonary edema, congestive heart failure, cardiac failure, and myocardial infarct. **Note:** Based on reports in the literature on tissue valves, there appears to be an increased incidence of leaflet calcification in patients under the age of 20. In this regard, animal research studies show that a high systemic calcium level can lead to early calcification. Furthermore, at least one published report describes an apparent causal relationship between the consumption of daily calcium supplements and early leaflet calcification in an adult. When feasible, repeated intravenous injections containing calcium should be avoided during the postoperative period; and excessive milk or dairy product consumption should be avoided in children. There are no clinical data presently available demonstrating increased resistance of Carpentier-Edwards bioprostheses to calcification as compared to other commercially available porcine valves. **Caution:** Federal (USA) law restricts this device to sale by or on the order of a physician. Baxter International Inc. is the owner of the following US patent which covers the products described herein: 4,106,129. Patent numbers in foreign countries supplied upon request. See package insert accompanying product for a full discussion of warnings, contraindications, complications and prescribing information. Carpentier-Edwards® is a registered trademark of Baxter International Inc.

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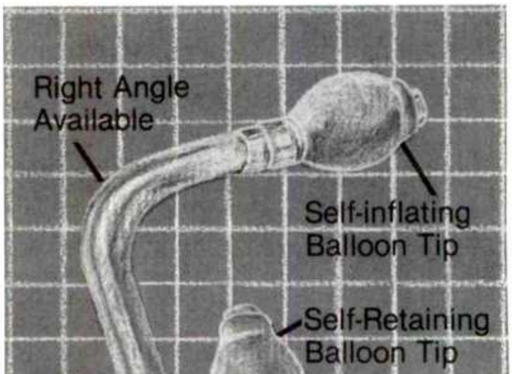
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Using ICD-9-CM and CPT in the Nineties

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In past years, cardiothoracic surgeons have seen little need to pay close attention to codes published in the *International Classification of Diseases 9th Clinical Modification* (ICD-9-CM) and *Physicians' Current Procedural Terminology* (CPT). Billing codes were the province of office personnel aided by computer systems.

Times have changed, however. In 1987 The Society of Thoracic Surgeons (STS), realizing the importance of reliable coding of operative procedures for our specialty, established the Ad Hoc Advisory Committee on CPT-4 Coding. The Committee was charged with evaluating all CPT codes for accuracy and relevance to state-of-the-art practices in cardiothoracic surgery.

Two members of the Committee, myself representing the STS and Dr Milton Weinberg representing the American Association of Thoracic Surgery (AATS), sit on the Physician's Advisory Committee of the American Medical Association's (AMA) Editorial Panel. The Advisory Committee, composed of physicians representing their specialty organizations, is charged with reviewing CPT codes and making recommendations on coding changes. An important assignment for each specialty's representatives is that of bringing its membership's suggestions to the attention of the Editorial Panel. The latter, composed of a group of AMA staffers, physicians, and insurance company representatives, ultimately makes all decisions on CPT revisions.

Last year, when Medicare mandated diagnostic ICD-9-CM coding on Medicare claim forms, the STS realized that the Ad Hoc CPT-4 Committee's charge was expanding, and it was reconstituted as the Committee on Nomenclature and Coding. It now stands as a permanent committee of the STS.

At this year's meeting of the AMA CPT Physician's Advisory Committee Meeting, AMA Trustee William Kacott, MD, observed that "Never before in our history has coding been so important." Accurate coding is one of the keys to physicians maintaining control over their practices in light of the ever-tightening regulations being exerted by the government and other third-party payers.

As we all know, cardiothoracic surgery did not fare well in the recent resource-based relative value study conducted by the Harvard School of Public Health for the Physician Payment Review Commission. All the evidence points to substantial decreases in Medicare/Medicaid reimbursement for cardiothoracic procedures. Other third-party payers are likely to follow suit.

In addition to general physician disinterest in coding, another reason for the adverse outcome associated with the resource-based relative value study was the imprecision with which the existing CPT has allowed us to report our work. For example, there are no specific codes for use of the internal mammary artery conduit, for redo cardiac operations, and for use of mechanical ventricular assist devices.

Most of us would agree that a redo coronary bypass operation can be difficult and time-consuming. To report this challenging procedure, we are reduced to using the generic modifier -22, which simply indicates "unusual services."

Code changes for redo operations and the internal mammary artery conduit were recommended by your Committee last year, but the Editorial Panel rejected these recommendations. We recently submitted a new set of arguments for these same changes with the hope that the Editorial Panel will better understand the rationale for new codes this time around.

In addition to proposing CPT code changes, the activities of the Committee have included: publication of *Using CPT for Cardiothoracic Reimbursement: A Manual for Surgeons and Insurance Billing Specialists*, an educational guide to help you and your office personnel use CPT better; organization of a coding workshop for physicians and office personnel presented at the STS's September 1990 interim meeting; serving as a resource for your questions on coding; and keeping physicians informed about appropriate coding practices.

To this last purpose, I wish to comment about the practice of "unbundling," ie, billing separately for the component parts of an operation. The Physician Payment Review Commission in concert with the Interspecialty Consensus Panel, the Health Care Financing Administration, and the CPT is proposing that surgical fees be global and should include immediate preoperative care, the surgical procedure itself, supportive procedures during the operation and immediate postoperative period, and routine postoperative care 90 days following the procedure. (This policy is in concert with the STS proposal on the cardiothoracic surgical bundle.) Although there are

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Doctor Levitsky is the Chairman of The Society of Thoracic Surgeons Ad Hoc Advisory Committee on Nomenclature and Coding; the members are Richard P. Anderson, O. Wayne Isom, Ellis L. Jones, C. Frederick Kittle, and Milton Weinberg, Jr. The Executive Secretary is Jeanne T. Fitzgerald.

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some exceptions with CPT starred procedures, the general rule is *one bill per operation*. Those who continue to "unbundle" may find themselves subject to nonreimbursement and possibly legal sanctions.

Precise coding has become very important in the smooth, everyday management of our practices. As Medicare and other payers put more controls on our affairs, we need rigorous coding practices to clearly portray the intensity and extended time commitments associated with our work. By doing so, we will position ourselves to receive appropriate reimbursement and provide accurate database input as governmental agencies and other third-party payers enter the realm of resource allocation (rationing) based on outcome management.

Assessing the Prognosis of Thymomas

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Thymomas have fascinated thoracic surgeons and pathologists since the original description of these neoplasms. There are few other human tumors that are associated with so many paraneoplastic manifestations, or that demonstrate such a breadth of microscopic appearances [1]. Prior studies on thymoma have centered largely on definitions of its pathological characteristics and on laboratory procedures designed to separate this lesion diagnostically from its histological mimics [2–6]. In addition, other analyses have begun to elucidate the immunological properties of constituent cells in thymomas, toward the end of better understanding their functional attributes [7, 8]. These assessments have had indirect benefits in estimating the prognosis of individual examples of these tumors, because they provide the pathologist with a definitive means of excluding differential diagnostic alternatives that have dissimilar behavioral characteristics. Such neoplasms include thymic carcinoid, thymic carcinoma, germ cell tumors of the mediastinum, and malignant lymphomas of the thymic region [1, 6].

Current work on the clinicopathological features of thymomas is focused more strongly on optimizing prognostication of these lesions. A body of literature accrued over the past 30 years suggests that, excluding tumors showing overt cytological anaplasia (which defines thymic *carcinomas* [9, 10]), there are few if any nonmacroscopic pathological findings that can be used to predict the behavior of thymomas [1, 3–6]. Nonetheless, recent reports have challenged the conclusion that the surgeon's gross impressions of invasiveness are paramount in prognostication. Masaoka and associates [11] found that microscopic staging of thymomas, reflecting various levels of neoplastic growth through the tumoral capsule, did indeed correlate with behavior. Moreover, Lewis and coworkers [6] observed a risk of recurrence that was proportional to the number of epithelial cells and the presence of cytologic atypia in such lesions, although these variables were admittedly inferior predictively to the presence or absence of macroscopic invasiveness.

See also page 534.

Perhaps the most novel, though somewhat contentious, publication in this area was that by Marino and Müller-Hermelink in 1985 [12]; it implied that separation of thymomas into "cortical" and "medullary" subgroups was prognostically important, with the former of these tumoral variants possessing greater biological aggressiveness. Definitions of the two groups are based on microscopic observations showing that epithelial cells of the normal thymic cortex are polygonal in shape and have small nucleoli, whereas those in the thymic medulla tend to be fusiform with evenly dispersed chromatin. Accordingly, neoplastic elements in cortical and medullary thymomas recapitulate these cytological features; "mixed" cortical-medullary tumors also are recognized. This histopathological approach is addressed in a paper by Pescarmona and associates in this issue of *The Annals* [13] in which 80 thymomas were categorized as cortical or medullary and staged using the criteria of Masaoka and colleagues. Multivariate statistical analysis of follow-up data in these cases appeared to support the utility of the Marino-Müller-Hermelink model, and Pescarmona and associates suggested that a prognostic "index" ([histologic type] × [stage]) should be employed by the pathologist in all examples of thymoma. Paraneoplastic conditions such as myasthenia were not found to be important prognostically, in accord with the observations of other recent publications [6, 14].

This information is of interest but, in my opinion, does not provide the final word on the subject under discussion here. The reasons for my dissent are threefold. First, it has proved difficult for surgical pathologists to agree with one another in the application of the Marino-Müller-Hermelink system. One is often unsure as to where the spectrum of cortical thymoma ends and that of mixed or medullary tumors begins. Despite having considerable experience with thymic neoplasms, I have personally been unable to confirm the usefulness of the cortical/medullary classification, and others have experienced the same problems [15]. Indeed, due to the variability of histological appearances in thymomas (even within the same tumor), it is likely that almost *any* microscopic classification system, including the traditional paradigm proposed by Bernatz and colleagues [16], will suffer from the same drawbacks. Second, I have serious questions on the soundness of the index introduced by Pescarmona and associates. It is based on the multiplicative score of two putatively interdependent variables, and therefore is imperfect from a statistical vantage point. Finally, Pescarmona and associates fail to state the number of medullary tumors that were examples of pure spindle-cell thymoma, a neoplastic subtype with a notoriously good prognosis [1, 6].

For the time being, I believe that staging procedures predicated on the degree of tumor invasion are still the best prognosticators for thymoma. Despite intense interest among pathologists in morphometric methods and flow cytometric evaluation of DNA content [17, 18], all of

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these procedures—like the Marino-Müller-Hermelink system—appear to be comparatively inferior or noncontributory techniques for predicting the biological behavior of these enigmatic lesions. It is certainly possible that one of many new technological advances being implemented today will prove to be the ultimate prognostic tool, but this eventuality remains to be seen.

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Traumatic Bronchial Rupture and Other Major Thoracic Injuries

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In this issue, Baumgartner and associates [1] report 9 cases of traumatic tracheobronchial disruption, collected from four trauma centers. They observed that early diagnosis and prompt repair by trained thoracic surgeons gave the best results. For example, all seven bronchoscopies done by thoracic surgeons accurately identified the lesions as compared with at least five of seven bronchoscopies by other physicians from which the findings were erroneous or incomplete. Although bronchial ruptures that caused total atelectases can be repaired with return of pulmonary function many years after the injury [2, 3], by far the best approach is immediate repair according to guidelines they outlined. It comes as no surprise that uncommon thoracic injuries receive suboptimum management by physicians who lack full training and experience in thoracic surgery.

What prompted the Baumgartner report? I suspect the cardiothoracic surgeons at Harbor/UCLA Medical Center, a major trauma center, may have been spurred to their report by late referrals of patients with tracheal and bronchial disruptions; they probably hoped to stimulate traumatologists of all disciplines toward earlier recognition and more prompt referral for definitive repair of tracheobronchial injuries.

See also page 569.

The facts presented by Baumgartner and associates indicate that thoracic surgeons have learned their lessons well, and one could therefore argue that publication of their report in *The Annals of Thoracic Surgery* is superfluous. Not so! There are important lessons about the practical management of thoracic trauma that are illustrated and implied, but not stated, in the Baumgartner report.

Five of 9 patients with traumatic tracheobronchial disruptions did not have the benefit of prompt, initial bronchoscopies by fully trained thoracic surgeons. This fact is a reflection of the existence of broadly trained traumatologists and emergency room medicine physicians as specialists in their own right. The Baumgartner report illustrates that bronchoscopy, once almost the exclusive turf of thoracic surgeons and selected otolaryngologists, became a generally used tool when the advent of flexible instruments simplified the procedure. With only a few little-heard warning cries [4], thoracic surgery quietly stepped aside. Today, too many candidates for examination by the American Board of Thoracic Surgery have had minimal experience with bronchoscopy. The Baumgartner report confirms the need for thoracic surgeons to remain competent, active endoscopists; we cannot rely on the findings of others.

What should be the thoracic surgical presence in trauma centers and emergency rooms? Theoretically we should care for *all* thoracic trauma. In practice, in most settings, thoracic surgeons cannot be instantly available to insert all necessary chest tubes at night and be fresh enough to carry out complex elective cardiac and thoracic operations in the morning. Thus, each trauma center and emergency room should have guidelines that encourage (and perhaps require) thoracic consultation under certain circumstances, eg, incomplete expansion of pneumothorax, widened mediastinum, penetrating injuries of the heart. In the interest of rendering the best available patient care, we must make ourselves readily available to emergency room and trauma center physicians despite the powerful competing forces of the operating room and consultations from cardiologists, pulmonologists, and gastroenterologists. As in the past, I hope that some thoracic surgeons will consider a career that emphasizes traumatology and regularly includes endoscopy. We must take positive steps toward retaining our role in endoscopy and the management of thoracic trauma. We must ensure that fully trained thoracic surgeons are regularly and readily involved with thoracic surgical problems in trauma victims.

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Relief of Tracheal Compression by Aortopexy

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We have performed aortopexy in 12 children with tracheal compression. Six infants had compression secondary to a vascular anomaly (group 1), and the other 6 had previous repair of esophageal atresia (group 2). Eleven of the 12 children are alive after a mean follow-up of 36 months. In group 1, 1 patient died and 3 patients (50%) experienced recurrent respiratory distress. Five infants sustained a major postoperative complication, and the average postoperative hospital stay was 25 days. In group 2, however, aortopexy was uniformly successful. There were no deaths, no postoperative complications, and no cases of postoperative respiratory distress, and the mean postoperative hospital stay was only 10 days. For children with reflex apnea after repair of esophageal atresia, aortopexy is lifesaving and can be performed with minimal morbidity and mortality. Great caution is indicated in children with tracheal compression from other causes.

(Ann Thorac Surg 1990;50:524–9)

The first known death resulting from mediastinal vascular compression was that of Jane Fordham in February 1761. Reporting to the Medical Society of London, David Bayford [1] explained that for 20 years, Fordham experienced severe dysphagia and odynophagia. At postmortem examination, he demonstrated that death was secondary to starvation caused by an anomalous right subclavian artery, which arose from the distal segment of the aortic arch and compressed the esophagus as it passed between it and the trachea.

Tracheal compression from mediastinal structures was not appreciated until 150 years later when Robert Gross noted a vascular ring during the postmortem examination of a 5-month-old baby who died from recurrent bouts of pneumonia. In 1945, Gross [2] reported the first successful relief of mediastinal tracheal compression by division of a vascular ring, and 3 years later, he and Neuhauser [3] described the first successful vascular "pexy" for mediastinal tracheal compression. In 1951, the same two authors [4] reported an extensive experience with aortopexy or vascular division, or both, in children with anomalous vascular compression of the trachea.

Tracheal compression can also occur from a dilated proximal esophagus in children with anastomotic stricture after repair of esophageal atresia. In 1974, Touloukian and colleagues [5] were the first to demonstrate such mediastinal crowding. Two years later, Filler and associates [6] and, separately, Benjamin and co-workers [7] successfully corrected this syndrome by aortopexy.

Thus there are two groups of children with mediastinal tracheal compression in whom aortopexy is potentially helpful. The first group consists of children with primary vascular anomalies encroaching on the trachea. The second group comprises children with esophageal anastomotic stricture from previous repair of esophageal atresia. Tracheal compression in these patients is a secondary phenomenon, which results from a dilated proximal esophageal pouch displacing the trachea anteriorly against the innominate artery. These patients frequently experience reflex apnea in association with feeding [8].

Material and Methods

Twelve infants with tracheal compression have undergone aortopexy at the Medical University of South Carolina over the past 5 years. A retrospective study was performed to establish the cause of tracheal compression, the presenting signs and symptoms, the modalities used in diagnosis, operative technique, and results.

The infants ranged in age from 2 weeks to 5 months, and half of them were male. Innominate artery compression was present in 4 patients, and vascular rings were diagnosed in 2 (group 1). Six patients had reflex apnea in association with a dilated esophageal segment proximal to an anastomotic stricture (group 2).

Presenting Signs and Symptoms

All of the infants in group 1 had chronic recurrent respiratory distress and pneumonia, but only 2 in group 2 experienced these problems. All of the patients in group 2 had a recurrent history of reflex apnea during feeding necessitating cardiorespiratory resuscitation compared with only half of the group 1 patients. Stridor was present in half of the patients in both groups. None of the group 1 infants experienced feeding difficulties, but 5 of the 6 infants in group 2 had dysphagia and regurgitation. One child with innominate artery compression demonstrated opisthotonic posturing. Table 1 summarizes the presenting signs and symptoms.

Preoperative Studies

The most challenging diagnostic aspect was confirmation of mediastinal tracheal compression as a cause of the observed symptoms. Excluding the more common diagnosis of gastroesophageal reflux was considered essential before aortopexy. Rigid bronchoscopy under general an-

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Table 1. Preoperative Signs and Symptoms[a]

Sign or Symptom	Frequency in Group 1 (n = 6)	Frequency in Group 2 (n = 6)	Frequency Overall (n = 12)
Reflex apnea	3 (50)	6 (100)	9 (75)
Chronic recurrent respiratory distress	6 (100)	2 (33)	8 (67)
Difficulty feeding	0 (0)	5 (83)	5 (42)
Stridor	3 (50)	3 (50)	6 (50)
Opisthotonic posturing	1 (17)	0 (0)	1 (8)

[a] Numbers in parentheses are percentages.

esthesia was performed in all patients. Anterior compression of the trachea by a pulsatile mass was seen uniformly (Fig 1). Complete tracheal collapse was noted in 5 of the 6 group 1 patients, but only 1 child with a history of esophageal atresia demonstrated this bronchoscopic finding. Thick airway secretions obstructing distal airways were noted in 5 of the 6 group 1 children but in only 1 child in group 2. In all 4 patients with innominate artery compression, it was possible to obliterate the right radial pulse by anterior displacement of the tip of the bronchoscope.

Half of the patients in group 1 and all in group 2 underwent preoperative esophagoscopy. The results of esophagoscopy in the 3 infants with vascular anomaly were inconsistent with gastroesophageal reflux. None of the group 2 patients had evidence of gastroesophageal reflux by esophagoscopy, and esophageal dilatation proximal to an anastomotic stricture was seen uniformly. Esophagography was helpful in the evaluation of 3 infants in group 1 by ruling out gastroesophageal reflux. Barium contrast studies of the esophagus confirmed the esophagoscopic findings in all group 2 patients (Figs 2, 3). Lateral fluoroscopic airway studies were helpful in 6 of the 9 children in whom they were performed. In such instances, anterior compression of the trachea was seen (Fig 4). On occasion, intermittent pulsatile occlusion of the tracheal lumen was noted on airway studies. Cardiac catheterization was employed in 1 patient with a vascular ring.

Operative Technique

The patient is positioned with the left shoulder elevated 30 to 45 degrees, and bronchoscopy is performed to confirm tracheal compression. Through a left anterior thoracotomy, partial thymectomy improves exposure and increases the effective cross-sectional area of the upper mediastinum. The apex of the left upper lobe is retracted inferiorly and posteriorly. A complete search for a vascular ring is conducted. A single row of interrupted monofilament sutures is placed from the arch of the aorta to the undersurface of the sternum and tied down securely so as to displace the arch anteriorly. Alternatively, the sutures can be passed through the sternum in small infants. Before closure of the left side of the chest, bronchoscopy is routinely repeated to demonstrate complete relief of tracheal compression.

Two technical details are worthy of mention. First, the bites into the aorta must be deep enough to purchase media and adventitia. Full-thickness bites, however, not only risk extensive hemorrhage but create the potential for postoperative aneurysm. Second, circumferential dissection of the aorta must be avoided. This dissection is unnecessary and disrupts the connective tissue between the posterior wall of the aorta and the anterior wall of the trachea. These attachments help to draw open the lumen of the trachea when aortopexy has been achieved.

Results

All 12 infants underwent aortic suspension with no intraoperative deaths. The aortopexy was extended onto the anterior surface of the innominate artery in 3 of the patients with innominate artery syndrome and in 3 from group 2. The aortopexy was extended onto the origin of the left common carotid artery in 1 patient with a vascular ring. Division of the innominate artery was necessary to relieve tracheal compression in 1 patient in group 1. No adverse neurological sequelae and no evidence of upper extremity claudication have been observed in this child. Two of the infants in group 1 underwent vascular ring division at the time of aortopexy and a third 8 weeks before aortopexy.

There was one early postoperative death: a 6-week-old infant with multiple cardiac and skeletal congenital anomalies died of sepsis. The other 11 patients are alive with 9 months' to 6 years' follow-up.

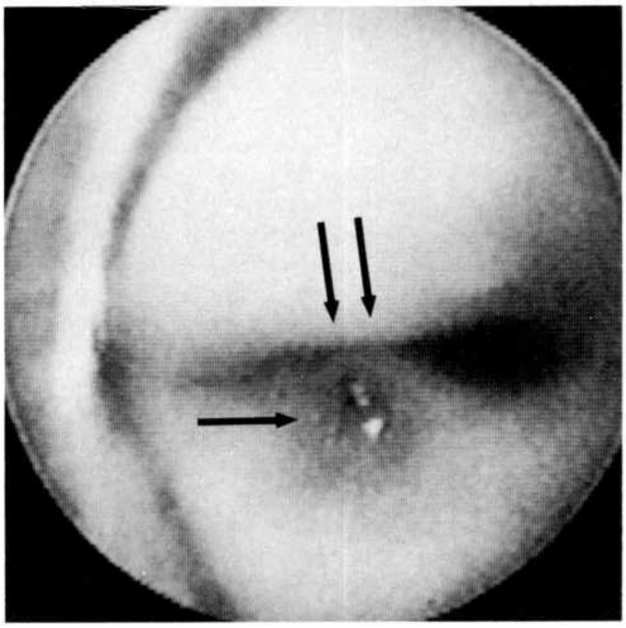

Fig 1. Preoperative bronchoscopic view in a 2-month-old child with reflex apnea who underwent repair of esophageal atresia and tracheoesophageal fistula at age 2 days. Note the remnant of the tracheoesophageal fistula (single arrow) *posterior to a completely collapsed trachea secondary to anterior compression by the innominate artery* (double arrows).

Intermediate and long-term results have been dependent on etiological group. In general, patients in group 1 fared poorly with half experiencing recurrent apnea and half requiring ventilatory support for more than 3 days postoperatively. All but 1 patient in group 1 had at least one major complication. The average postoperative hospital stay in this group was 25 days. In contrast, the patients in group 2 had no episodes of apnea postoperatively; All were extubated shortly after operation, and there were no complications. The average postoperative hospital stay in this group was 10 days. Table 2 summarizes the follow-up data.

Comment

A sufficient number of infants with vascular compression of the trachea have been seen at the Medical University of South Carolina to convince us that the syndrome exists. Aortopexy has been most effective in children in whom this problem develops because of anastomotic stricture of the esophagus. However, it is clear that two distinct disease processes can lead to severe anterior tracheal compression. Although the separation of these two groups has not previously been emphasized, our experience has established clear differences in both clinical presentation and response to aortopexy.

Recurrent bouts of pneumonia and respiratory distress predominate in children with vascular anomaly. This pattern of presentation was noted early by Gross and Neuhauser [2–4]. Conversely, reflex apnea is the typical clinical presentation in children with tracheal compromise from a dilated proximal esophagus. The term *reflex apnea* was popularized in 1963 by Fearon and Shortreed [9]. They observed cessation of respirations in a small number of children with a history of apnea when the area of tracheal compression was impinged upon by the tip of a bronchoscope. It was subsequently shown that reflex apnea in children with vascular anomaly is rare [10]. Reflex apnea was present in only one third of our infants with vascular anomaly, but it was uniformly seen in children requiring aortopexy after repair of esophageal atresia.

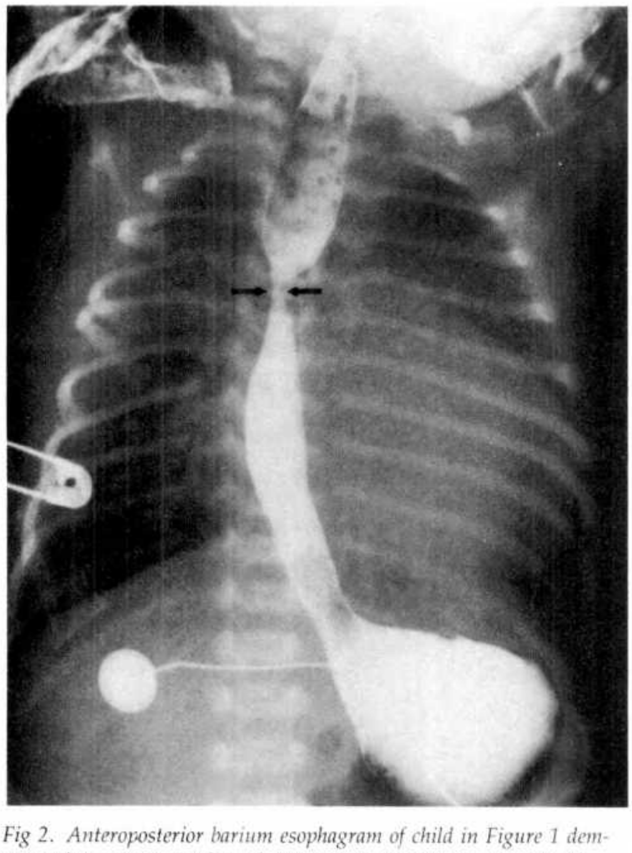

Fig 2. Anteroposterior barium esophagram of child in Figure 1 demonstrating narrow esophageal anastomotic stricture (arrows) *with extensive dilatation of proximal segment.*

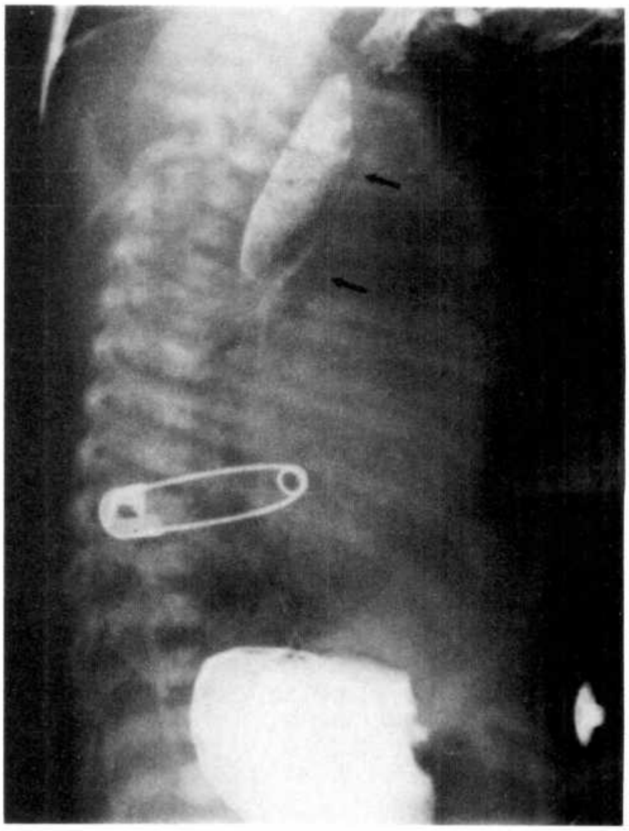

Fig 3. Lateral barium esophagram of child depicted in Figures 1 and 2 before aortopexy. Note the normal segments of trachea above and below the area of marked narrowing (between arrows*) caused by forward displacement of the airway into the aortic arch and innominate artery by the dilated proximal esophagus.*

Another contrast between the two groups is the finding of difficulty with feeding. The problem is common in children requiring aortopexy after repair of esophageal atresia. The proximal esophageal pouch dilates because of anastomotic stricture. Such association of respiratory distress during feeding is rare in children with tracheal compression from mediastinal vascular malformations. One exception is dysphagia lusoria, and in such patients, concomitant aortopexy may be appropriate if tracheal compression persists after correction of the vascular lesion [11, 12].

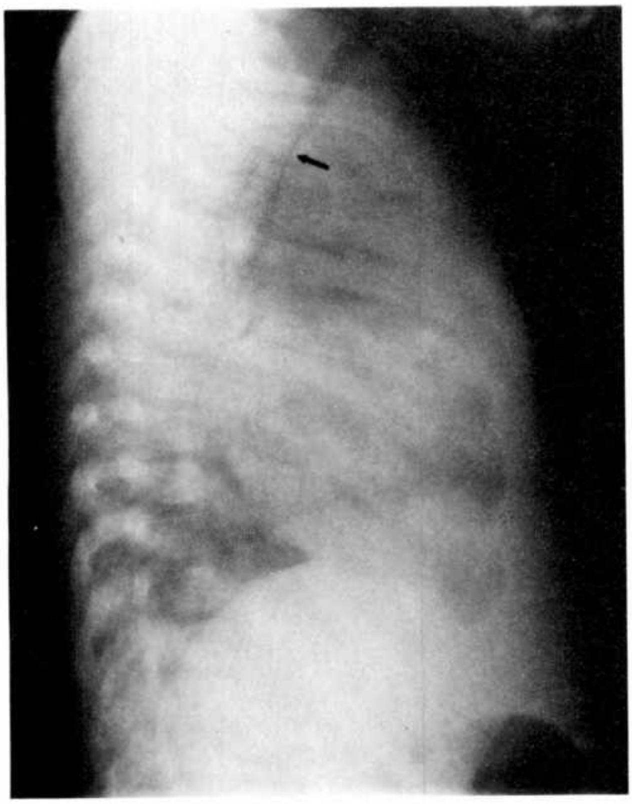

Fig 4. Lateral tracheobronchogram of child in Figures 1 through 3 demonstrating marked narrowing of the trachea due to anterior compression (arrow).

Gross and Neuhauser [3] emphasized the finding of opisthotonos in the first child to undergo aortopexy for innominate artery compression, and Gross [4] reiterated its importance in a much larger experience 10 years later. Berdon and co-workers [13] isolated stridor as an important diagnostic finding in children with innominate artery compression. Both stridor and opisthotonic posturing were inconsistent findings in our study.

The diagnosis of tracheal compression syndrome is best made by bronchoscopy. In children with a clear history of reflex apnea, a pulsatile mass compressing the anterior mediastinal trachea is pathognomonic and represents an absolute indication for aortopexy. In our experience, barium studies of the esophagus are also helpful to exclude the diagnosis of gastroesophageal reflux. It has long been appreciated that gastroesophageal reflux is common after repair of esophageal atresia [14, 15]. Furthermore, apneic spells in children with suspected vascular compression may be more frequently due to aspiration from gastroesophageal reflux than reflex apnea [8]. For these reasons, we believe that upper gastrointestinal barium studies are helpful in establishing which children are candidates for aortopexy.

Rigid esophagoscopy is also helpful in identifying stricture and proximal esophageal dilatation in infants with a history of esophageal atresia. Berdon and associates [13] emphasized the value of the lateral tracheobronchogram. In our series, curvilinear anterior indentation of the trachea was present in 75% of the patients; however, its presence was not predictive of surgical success. Cardiac catheterization was helpful in 1 of our children but is not considered routine, even when primary vascular anomaly is suspected.

The response to aortopexy was generally poor in our patients with suspected vascular anomaly, whereas the patients with a history of esophageal atresia responded well. We believe several reasons exist for these results. Reflex apnea, the predominant symptom in group 2, implies a comparatively sound, albeit collapsible, tracheobronchial tree. The more indolent pattern of recurrent bouts of pneumonia and chronic respiratory distress seen in children with suspected vascular abnormality suggests a less anatomically correctable cause for airway compromise. Bradham and colleagues [16] suggested that inhibition of normal in utero tracheal development can occur secondary to compression from mediastinal vascular anomalies. Extensive retardation in tracheal development by anomalous vascular compression has been hypothesized by others [9, 16, 17]. Thus, irreversible tracheal disease might explain poor results in children with vascular anomaly.

Although the association of defective tracheal structure with esophageal atresia has been noted [18, 19], Benjamin and co-workers [7] pointed out the importance of other factors (mediastinal crowding, in particular) that contribute to tracheal collapse in these children. Probably because of more normal tracheal development, children with mediastinal crowding after repair of esophageal atresia respond better to aortopexy than children with congenital vascular anomaly. However, because of the progressive defect in tracheal development that accompanies vascular compression, it is appropriate to consider tracheal suspension at the time of division of a vascular ring.

A second reason for superior results in children with anastomotic stricture is the distinct nature of the diagnosis. It has been our experience that the contribution of tracheal compression from vascular anomalies often is not obvious. These children tend to have multiple factors leading to airway compromise and respiratory distress.

Table 2. Summary of Follow-up Data[a]

Variable	Group 1 (n = 6)	Group 2 (n = 6)	Overall (n = 12)
Deaths	1 (17)	0 (0)	1 (8)
Recurrent apnea	3 (50)	0 (0)	3 (25)
Postoperative ventilator dependence	3 (50)	0 (0)	3 (25)
Major complication	5 (83)	0 (0)	5 (42)
Mean postoperative hospital stay (d)	25	10	17.5

[a] Numbers in parentheses are percentages.

This point is best made by looking at the operative failures in our series. The single death occurred in an infant with multiple anomalies and chronic ventilatory insufficiency from birth. A Nissen fundoplication was performed for gastroesophageal reflux, with little improvement in respiratory status. Bronchoscopy demonstrated tracheobronchitis, impacted airway secretions, and a floppy trachea, which collapsed completely at the level of a pulsatile anterior mass. Anterior displacement of the mass by the tip of the bronchoscope obliterated the right radial pulse. Although a temporary improvement in the respiratory condition was seen after aortopexy, the child died of sepsis 3 weeks postoperatively. Another patient also had presumed innominate artery compression syndrome, but after aortopexy, persistent respiratory distress and apnea were found to be secondary to Dandy-Walker syndrome with bilateral vocal cord paralysis. A tracheostomy was ultimately required. The third patient in whom aortopexy was considered a failure had undergone vascular ring division 8 weeks before aortopexy. Bronchoscopy before tracheal suspension demonstrated total tracheal collapse on exhalation with inspissated airway secretions. Aortopexy led to no improvement in respiratory status. Subsequent placement of an extraluminal tracheal stent has achieved progressive improvement. We believe that failure in the first 2 patients was due to diagnostic error. The third child, however, may represent a situation in which aortopexy was considered too late. Had aortopexy been performed concomitant with the original vascular ring division, reversal or at least arrest of the developmental tracheal effect might have been possible.

Our experience underscores the life-threatening nature of reflex apnea. The syndrome can be expected during follow-up of large groups of infants who have undergone repair of esophageal atresia. An indolent form of chronic recurrent respiratory distress is more commonly seen in children with suspected mediastinal vascular compression of the trachea. Although aortopexy has a place in the management of both groups, results are much better in the former, and we are enthusiastic about recommending the procedure in that setting. Care must be taken in patients with vascular compression to complete a careful diagnostic workup and to rule out irreversible tracheomalacia. If a complete vascular ring is divided, concomitant aortopexy may be appropriate. If there is no vascular ring and only anterior innominate compression, aortopexy can be performed with guarded optimism.

We thank Megan Othersen for her creative editorial assistance and Diane Robinson for her patient preparation of the manuscript.

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INVITED COMMENTARY

This paper from the pediatric surgery service of the Medical University of South Carolina is important because it documents two distinct causes of vascular tracheal compression in babies: (1) *vascular anomalies* that may cause external compression and symptomatic respiratory distress and (2) compression of the trachea from a *dilated esophageal pouch* that is trapped in scar tissue after operative repair of tracheoesophageal fistula.

Respiratory distress secondary to vascular compression was emphasized in the middle 1940s by Dr Robert Gross of the Children's Hospital of Boston, who also introduced the suspension aortopexy to take the pressure of the aortic arch off of the trachea. He did not recognize that there is frequently an associated abnormality of the tracheal wall, a form of chondromalacia, that is closely related to the vascular anomaly and may decrease the chances for immediate relief of symptoms after the aortopexy. The operation for this group of patients has remained controversial because of the difficulty in selecting those patients who truly will benefit from suspension of the aortic arch or of the innominate vessel if it were the offending vessel. Doctor Clevenger and colleagues have documented that this is a small group of patients and that they can be identified by careful bronchoscopy and esophagoscopy, both before and after the operative procedure and under the same anesthesia. Not infrequently, these children will require months before there is further stabilization of the weak wall of the adjacent trachea but, in time, these children do well.

The second cause, tracheal compression after repair of tracheoesophageal fistula, is very unusual in my experience, and it is a complication only if there is an esophageal stricture at the site of the anastomosis. I am not sure why Clevenger and associates have so many cases. Strictures occur in less than 10% of cases. They were more common when the older double-layer Height anastomosis was used than with the newer single-layer anastomosis.

The first group (vascular anomalies) is much larger, and Clevenger and associates appropriately urge great caution in treating tracheal symptoms due to assumed vascular compression. Aortopexy should not be considered unless severe compression has been well documented by careful endoscopy. This syndrome is extremely rare. Suspension cures only a modest percentage of these patients because intrinsic tracheal malacia is often the primary cause of the collapse.

This paper is a well-balanced presentation and a very helpful delineation of these two groups of patients.

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Selective Monitoring of Patients With Suspected Blunt Cardiac Injury

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Blunt chest trauma can result in cardiac injury with consequent dysrhythmias, valve malfunction, or frank rupture. Typically, patients with blunt chest trauma and suspected cardiac injury have required cardiac monitoring for 48 to 72 hours. Predicting which patients with blunt chest trauma are not at risk for cardiac complications would obviate many patient-hours of monitoring in the intensive care unit. This series examines the sensitivity of two-dimensional surface echocardiography in predicting cardiac complications. Over a 24-month period, 115 patients were admitted with blunt chest trauma and prospectively evaluated for cardiac injury with admission electrocardiograms, serial creatine kinase isoenzyme studies, and two-dimensional echocardiography. Thirty-one patients (27%) had abnormal two-dimensional echocardiograms. In 8 (25.8%) of these patients, cardiac complications requiring treatment developed. Eighty-four patients (73%) had normal two-dimensional echocardiograms, and a cardiac complication requiring treatment developed in only 1 (1.2%) of them. Of the 9 patients who required treatment of cardiac complications, 3 had normal admission electrocardiograms and only 1 had elevated levels of the myocardial-specific isoenzymes of creatine kinase. We believe two-dimensional echocardiography is a sensitive test for evaluating cardiac injury resulting from blunt chest trauma and is helpful in selecting those patients who require monitoring in the intensive care unit.

(Ann Thorac Surg 1990;50:530–3)

Blunt chest trauma can result in a broad spectrum of cardiac complications including supraventricular and ventricular arrhythmias, valve malfunction, cardiac chamber disruption, and pericardial tamponade. Patients suspected of blunt cardiac injury have required continuous hemodynamic and electrocardiographic monitoring in the intensive care unit (ICU) until cardiac injury can be excluded by a variety of diagnostic methods. Objective evaluation of possible blunt cardiac injury has included the electrocardiogram (ECG), serial studies of the myocardial-specific isoenzyme of creatine kinase (CK-MB), and, more recently, two-dimensional (2-D) surface echocardiography and gated ventricular angiography. Continuous cardiac monitoring in the ICU involves the use of valuable resources in the case of patients in whom clinically significant cardiac sequelae fail to develop. Predicting which patients sustaining blunt chest trauma are not at risk for cardiac complications would safely obviate many ICU admissions and many patient-hours of ICU monitoring.

Material and Methods

Over a 24-month period, 115 patients were admitted to St. Joseph Mercy Hospital in Ann Arbor, MI, with blunt thoracic trauma, and all were prospectively evaluated for blunt cardiac injury with admission ECGs, serial CK-MB analyses, and 2-D echocardiography. There were 75 male and 40 female patients ranging in age from 10 to 75 years with an average age of 37 years. The majority of patients had multiple injuries after motor vehicle accidents. An Injury Severity Score was calculated for each patient [1]. The average score for all patients was 16.2, and that for patients with cardiac sequelae was 26.4. When possible, a history of prior coronary or valvar heart disease was obtained. Thirteen percent (15 patients) of all patients and 2 (22%) of the 9 patients who required treatment of cardiac sequelae had a known history of heart disease. All patients were monitored in an ICU until blunt cardiac injury was excluded or until the other injuries allowed transfer to a general care bed.

Creatine kinase-MB level was assayed in blood samples drawn on admission and every 8 hours for a 24-hour period. The assay was considered positive if the MB fraction comprised 5% or more of the total CK. Two-dimensional echocardiography was performed in the ICU on the day of admission when possible or on the first morning after a nighttime admission. Any abnormality on 2-D echocardiography including valve malfunction, wall motion abnormalities, and pericardial effusions was considered positive. Electrocardiograms were considered abnormal if any atrial or ventricular irritability was demonstrated or if aberrant conduction existed. Sinus tachycardia was considered normal in a traumatized patient.

Presented at the Thirty-sixth Annual Meeting of the Southern Thoracic Surgical Association, Scottsdale, AZ, Nov 9–11, 1989.

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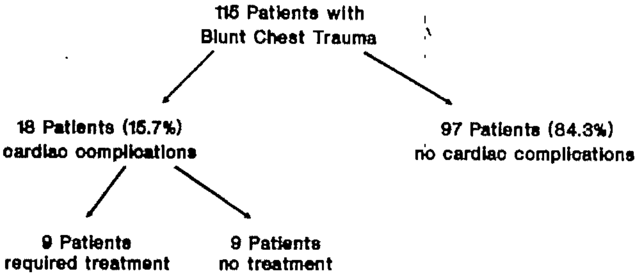

Fig 1. Distribution of patients with blunt chest trauma.

Table 2. Results of Serial Studies of Creatine Kinase-MB

Patient Category	Elevated[a]	Normal	Not Done
All (n = 115)	1	107	7
Without cardiac complications (n = 97)	0	91	6
With cardiac complications (n = 18)	1	16	1
Requiring treatment (n = 9)	1	8	0

[a] Elevated means the MB fraction is greater than or equal to 5% of the total creatine kinase.

Results

Of the 115 patients admitted with blunt thoracic trauma, 18 (15.7%) experienced cardiac complications ranging from occasional premature ventricular contractions to cardiac arrest. The other 97 patients (84.3%) had no cardiac sequelae.

Of the 18 patients in whom cardiac complications developed, 9 had serious sequelae that required treatment. Treatment included antiarrhythmic drugs for supraventricular and ventricular tachyarrhythmias and pericardiocentesis for tamponade. The remaining 9 patients had clinically insignificant cardiac sequelae, such as occasional premature ventricular contractions, which did not require treatment (Fig 1).

Admission ECGs were obtained for 109 of the 115 patients and were abnormal in 54 patients (49.5%). Of the 18 patients who had cardiac complications, 5 had normal admission ECGs, including 3 of the 9 who required treatment for the complications (Table 1).

Serial CK-MB studies were done for 108 patients. Using the 5% MB fraction as the threshold of positivity, only 1 patient had elevated levels of CK-MB, a 37-year-old man with no known history of coronary artery disease. Of the 9 patients with clinically significant cardiac sequelae, 8 (89%) did not have elevated CK-MB levels (Table 2).

Two-dimensional echocardiography was performed on all 115 patients. Thirty-one patients (27%) had abnormal 2-D echocardiograms and 84 patients (73%) had normal results. Of the 31 patients with abnormal results, 8 (25.8%) had cardiac complications requiring treatment. In contrast, of the 84 patients with normal echocardiography, only 1 patient (1.2%) had a cardiac complication requiring treatment: a 56-year-old man was given digoxin in the emergency room for transient atrial fibrillation (Table 3). By χ^2 analysis, these two groups are significantly different ($p < 0.00001$). Furthermore, the sensitivity of echocardiography in predicting a clinical problem is 0.258 and the specificity, 0.988, thus indicating that if the echocardiogram is normal, cardiac complications are very unlikely.

Comment

The clinical significance of cardiac injury after blunt thoracic trauma is well recognized; however, the method of evaluation is controversial. Several institutions have demonstrated the insensitivity of electrocardiography [2-4]. Serial CK-MB studies have been recommended for the evaluation of blunt cardiac injury [5, 6]. However, negative results of such studies do not exclude blunt cardiac injury, and their routine use is questionable [7, 8]. Routine cardiac monitoring in an ICU of all patients with suspected blunt cardiac trauma will detect serious arrhythmias, but it is expensive and uses valuable resources. Radionuclide angiography has shown promise as a sensitive test and warrants further investigation [2, 3]. Frazee and colleagues [4] have demonstrated the usefulness of 2-D echocardiography in the evaluation of blunt cardiac injury, and our experience with this method is similar to theirs.

Given the predictive value of 2-D echocardiography, patients can be selectively monitored for blunt cardiac injury. If the patient requires ICU monitoring for associated injuries, regardless of the possible blunt cardiac injury, then a baseline ECG should be obtained and the patient monitored as long as the other injuries make it necessary. However, the patient who does not have associated injuries requiring ICU monitoring should un-

Table 1. Results of Electrocardiography

Patient Category	Abnormal	Normal[a]	Not Done
All (n = 115)	54	55	6
Without cardiac complications (n = 97)	42	50	5
With cardiac complications (n = 18)	12	5	1
Requiring treatment (n = 9)	6	3	0

[a] Sinus tachycardia is considered normal.

Table 3. Results of Two-Dimensional Echocardiography

Patient Category	Abnormal	Normal	Not Done
All (n = 115)	31	84	0
Without cardiac complications (n = 97)	13	75	0
With cardiac complications (n = 18)	12	6	0
Requiring treatment (n = 9)	8	1[a]	0

[a] This patient had transient atrial fibrillation in the emergency room.

Table 4. Clinical Features of Patients With Cardiac Complications After Blunt Chest Trauma

Patient No.	Sex	Age (y)	Injury Severity Score	Admission Electrocardiogram	CK-MB Study	Echocardiogram	Complication (Treatment)
1	F	14	23	Sinus tachycardia	–	Severe LV dysfunction	Ventricular tachycardia (lidocaine hydrochloride), cardiogenic shock
2	M	20	50	Sinus tachycardia, nonspecific ST-T wave changes	–	Mild regional LV dysfunction, decreased septal contractility	Supraventricular tachycardia (verapamil hydrochloride)
3	F	67	50	Sinus tachycardia, ? inferior MI	–	Severe LV hypertrophy, IHSS, MR, LA	Atrial fibrillation (amiodarone hydrochloride)
4	M	20	35	Sinus tachycardia	–	Large pericardial effusion	Tamponade (pericardiocentesis)
5	M	61	19	ST-T wave changes	–	Pericardial effusion	Frequent PVCs (lidocaine)
6	M	16	35	Sinus tachycardia, ST-T wave changes	–	Severe LV dysfunction	Cardiac arrest (resuscitation)
7	M	37	17	Anterolateral MI	+	Apical thrombus, akinetic anteroseptal wall, moderate regional LV dysfunction	Acute MI (PTCA 6 wk after injury)
8	M	47	18	Sinus tachycardia	–	Pericardial effusion	Atrial flutter (digoxin, procainamide hydrochloride)
9	M	56	10	Atrial fibrillation	–	Normal (technically difficult)	Transient atrial fibrillation (digoxin in emergency room)

CK-MB = myocardial-specific isoenzyme of creatine kinase; IHSS = idiopathic hypertrophic subaortic stenosis; LA =left atrium; LV = left ventricular; MI = myocardial infarction; MR = mitral regurgitation; PTCA = percutaneous transluminal coronary angioplasty; PVC = premature ventricular contraction.

dergo 2-D echocardiography. A normal 2-D echocardiogram would allow the patient to safely forgo ICU admission or further evaluation for blunt cardiac injury. An abnormal echocardiogram, however, would indicate a patient at risk for the development of cardiac sequelae and therefore the patient should be monitored for 48 to 72 hours. Table 4 provides a summary of the data on the 9 patients requiring treatment.

In conclusion, routine cardiac monitoring of the patient with blunt chest trauma and suspected cardiac injury is expensive and uses valuable resources. Two-dimensional echocardiography has proved to be a sensitive test for evaluating blunt cardiac injury and is helpful in selecting those patients who require ICU monitoring.

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DISCUSSION

DR JAMES W. PATE (Memphis, TN): This excellent paper addresses a very important, practical clinical question. I think the most interesting thing is the question it raises. As the Presidential Address pointed out yesterday, the question is frequently more important than the data that arise from it.

The relevant question here is not how to diagnose cardiac injuries. Tamponade, rupture of the heart, valve injury, coronary occlusion, and laceration are all diagnosed by different techniques. The question is not how to diagnose cardiac contusions. Autopsy studies of patients with blunt chest injuries show that cardiac contusions can be found in up to 75% of patients in some series and usually have no clinical importance. All of us who have seen the heart after cardiopulmonary resuscitation, either at operation or autopsy, know that 100% of those hearts have major cardiac contusion, and yet that is of little, if any, clinical import. Reif and associates phrase the question in terms of patients who require monitoring. I do not think that this is quite the right question.

My associates and I have worded the question in this way: Which patient with no other evidence of cardiac injury should be electronically monitored for arrhythmias? Arrhythmia is the problem about which we are most concerned. Which patients should have special management for general anesthesia?

We reviewed 2,063 admissions. Of these patients, 1,110 had blunt injuries and 140 of them had evidence of chest injury.

In a prospective study, we did a 12-lead ECG on admission and daily, and continued electronic monitoring for 72 hours. Studies of CK-MB were done on admission and every 6 hours. The GVA was done as soon as feasible. Two-dimensional echocardiography was performed, and cardiac output and pulmonary and systemic vascular resistance were calculated. In patients seen later, Holter monitoring was done for 72 hours.

For patients who had, on the Holter monitor, premature ventricular contractions of more than five consecutive beats, there was a good correlation with the CK-MB studies. A very important observation about the CK-MB studies is that in 75% of the patients with elevated levels of CK-MB, the elevation was noted *only* on admission or *only* at the 6-hour period. Unless both an admission study and a 6-hour CK-MB study are done, the elevation may be missed.

The CK-MB studies correlate well with the hemodynamic abnormalities but not with echocardiography. The Holter monitoring correlates with CK-MB analysis and also indicates that the arrhythmias appear 2 to 3 days after injury.

Of the last 11 patients who had premature ventricular contractions of more than 100 beats per 24 hours in an ICU with good nurses and state-of-the-art equipment, only 2 had their premature ventricular contractions observed by the electronic equipment or the nurses. So electronic monitoring itself is not the gold standard.

Dr Reif, in the present series, when were the CK-MB studies done, and how did changes correlate with the clinical findings?

DR REIF: Our samples for CK-MB studies were drawn on admission and every 8 hours for a 24-hour period. Using 5% or greater as the cutoff, we had only 1 patient in whom the level of this isoenzyme was elevated. There are some investigators, such as Frazee and Mucha at the Mayo Clinic, who have a much lower threshold and use trace or 1% MB fraction as a positive.

DR WILLIAM A. COOK (North Andover, MA): Now that Mr Braimbridge has taught us to ask questions instead of offering discussion, I will ask two questions. First, do you think it is appropriate to allow fiscal considerations to be the determinant of quality of care for your patients? Second, do you really think that monitoring the ECG is the sole reason for having a patient in the ICU, particularly a patient who has a severe blunt injury of the chest?

DR REIF: In answer to your first question, as long as the quality of care is not compromised, I think that cost considerations are valid. Second, certainly the only reason to have a patient in the ICU is not for continuous cardiac monitoring. Usually these patients have multiple injuries and are admitted to that unit for other reasons also.

DR HOOSHANG BOLOOKI (Miami, FL): I have a case history to report. A young patient was admitted to Jackson Memorial Hospital in Miami because of chest trauma. In the emergency room he was in circulatory shock and had an exploratory thoracotomy. There were no cardiac injuries. In the ICU he remained hypotensive and was in severe cardiac failure. Echocardiography showed an ejection fraction of 0.10 to 0.12. A diagnosis of severe cardiac dysfunction was made.

An intraaortic balloon pump was used as a temporary assist. The patient underwent cardiac catheterization while on balloon assist. All coronary arteries were patent. Cardiac function was followed with daily echocardiograms. On the third day, the ejection fraction had improved to about 0.35. At that time, the balloon pump was discontinued.

This is the only patient of whom I am aware who has had balloon pumping for cardiac trauma without involvement of the coronary arteries. This experience also shows that the echocardiogram is a valuable tool in patients with chest trauma and can be lifesaving.

DR SAMUEL H. SADOW (Palm Beach Gardens, FL): I enjoyed your paper very much. I have two comments and a question. Having always been taught that the presentation of myocardial contusion can be a subtle spectrum, from a simple dysrhythmia to a full blowout of a valve or ventricular chamber, I believe that the best diagnostic tool we have is a high index of suspicion because of the mechanism of injury. Second, the diagnosis of myocardial contusion based purely on elevations of CK-MB may be unreliable when the CK level exceeds 3,000 IU from massive soft-tissue trauma because under those conditions, the MB fraction may be spuriously elevated. My question is, What are your criteria for monitoring patients with possible myocardial contusion who are not obviously desperately ill? Do you monitor all of them, and for how long is monitoring continued?

DR REIF: I thank Dr Bolooki for sharing his interesting case report. We did not have any patient who required an intraaortic balloon pump.

Dr Sadow, as for the CK-MB study giving a false-positive result with other injuries (severe skeletal muscle or tongue trauma), it is true that the isoenzyme study can lead to a false-positive result, but I would be more concerned about a false-negative CK-MB study in which the MB fraction came back negative but the patient was still at risk of a cardiac complication. Your follow-up question concerned which patients we select for monitoring, and the answer is any patient who has had a rapid deceleration injury or a mechanism of injury consisting of a direct blow to the sternum. Most of our patients were involved in motor vehicle accidents and had multiple injuries.

Analysis of Prognostic Factors and Clinicopathological Staging of Thymoma

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The prognostic value of four clinical variables (age and sex of patients, association with myasthenia gravis, and clinical stage) and histological type was analyzed in 83 consecutive patients with thymoma, histologically classified as cortical, medullary, and mixed. Age, sex, and association with myasthenia gravis did not prove to represent significant prognostic factors; clinical stage and histological type, on the contrary, had a highly significant prognostic value ($p < 0.001$). A model of clinicopathological staging, based on both clinical stage and histological type, in which three major prognostic groups are considered is proposed. The degree of significance of this model is higher ($p < 0.0001$) than that of clinical stage and histological type considered individually; its validity is further supported by the results of multivariate analysis according to the Cox regression model ($p = 0.0001$). We think it represents a prognostically valuable approach to the problem of management of thymoma.

(Ann Thorac Surg 1990;50:534–8)

It is well known that epithelial tumors of the thymus, thymoma and thymic carcinoma, are characterized by a peculiar histological polymorphism and a heterogeneity of biological and clinical behavior [1]. Histology is a prominent prognostic factor in patients with thymic carcinomas, which are very unusual epithelial neoplasms with obvious malignant cytological characteristics [2]. However, the prognostic value of histological typing of thymomas, which lack clear-cut cytological atypias [3], is still controversial [4–6]. We [7, 8] recently reported that the classification of thymomas as cortical, medullary, and mixed types, as proposed by Marino and Müller-Hermelink [9], allows reliable clinicopathological correlations and has important prognostic implications.

For editorial comment, see page 521.

In the present study, we analyzed the prognostic value of a few clinical variables (age and sex of patients, association with myasthenia gravis [MG], and extent of the tumor as defined by clinical stage) in a series of 83 consecutive patients with thymoma, histologically classified as cortical, medullary, or mixed. The purpose of our study was to determine a model of clinicopathological staging of thymoma that might be of value from prognostic and therapeutic standpoints.

Material and Methods

Eighty-three consecutive patients with thymoma treated surgically at the Department of Thoracic Surgery of the University of Rome "La Sapienza" between 1965 and 1989 were considered in this study. For each patient, clinical records (age, sex, association with MG, and clinical stage) and precise follow-up data were available. All histological material was reviewed by three of us (E.P., C.D.B., and L.P.R.) on routinely stained slides; only tumors fulfilling the diagnostic criteria of thymoma [1–3] were included and histologically classified according to Marino and Müller-Hermelink [9] as cortical, medullary, or mixed type. Tumors that displayed clear-cut malignant histological features, ie, thymic carcinoma, were excluded.

The degree of invasiveness of each tumor was evaluated according to the clinical staging procedure proposed by Masaoka and colleagues [10]. Stage I tumors were macroscopically encapsulated with no microscopically detectable capsular invasion; stage II thymomas featured microscopic invasion into the capsule or macroscopic invasion of mediastinal fatty tissue or mediastinal pleura; stage III tumors displayed macroscopic invasion of the neighboring structures (pericardium, great vessels, or lung); and stage IV thymomas had pleural or pericardial dissemination, lymphogenous or hematogenous metastasis, or both dissemination and metastasis.

To assess their prognostic value, the following factors were analyzed, first individually and then in a multivariate analysis: age (20 to 40 years, 40 through 60 years, and more than 60 years), sex, association with MG, clinical stage, and histological type. Operative deaths were included, and deaths related to MG alone were excluded. The statistical analysis was done with the χ^2 test. Survival curves were plotted according to Kaplan and Meier [11] and analyzed using the log-rank [12] and Lee-Desu tests [13]. These analyses were performed with the SPSS-X

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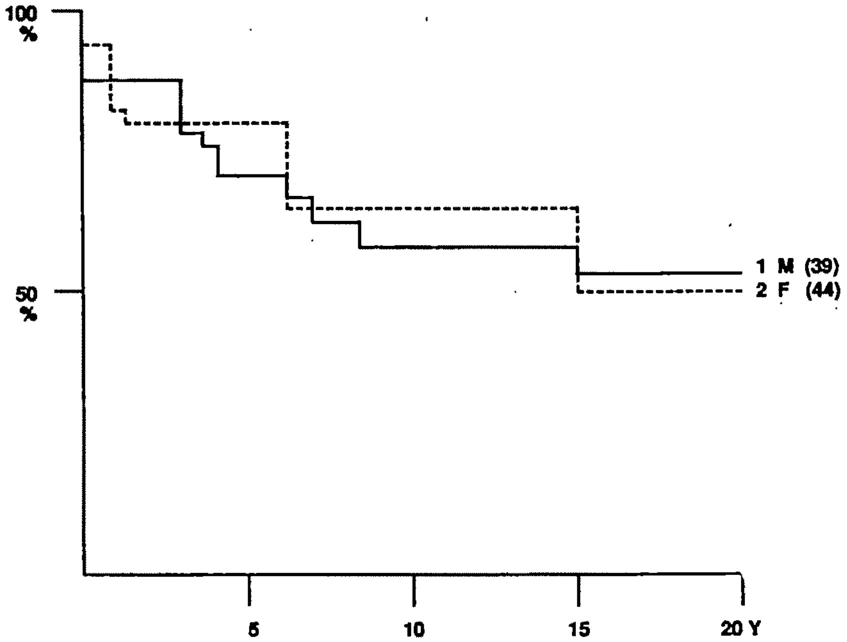

Fig 1. Actuarial survival curves based on sex of patients ($p = 0.72$*).*

package [14]. The multivariate analysis was done using the Cox regression model [15] with a stepwise procedure (BMDP package).

Results

Of the 83 patients, 44 were women and 39 were men. Age ranged between 21 and 83 years (mean age, 48.6 years). Myasthenia gravis was present in 25 patients (30%) (12 with mixed, 11 with cortical, and 2 with medullary histology). Histologically, 34 patients (41%) had cortical, 13 (16%) had medullary, and 36 (43%) had mixed type. Clinically, 23 patients (28%) were in stage I, 26 (31%) were in stage II, 26 (31%) were in stage III, and 8 (10%) were in stage IV. A large majority of the patients with cortical histology (27 or 79%) were in stage III or IV, whereas most of those with medullary histology (11 or 85%) were in stage I; patients with mixed histology showed an intermediate degree of invasiveness (29 or 81% in stage I or II).

The analysis of the survival curves showed no significant differences when sex ($p = 0.72$) (Fig 1), age ($p = 0.94$) (Fig 2), and association with MG ($p = 0.82$) (Fig 3) were considered individually. Clinical stage ($p < 0.001$) (Fig 4) and histological type ($p < 0.001$) (Fig 5), on the contrary, proved to be highly significant prognostic factors. Consequently, actuarial survival curves of a model of clinicopathological staging based on both clinical stage and histological type were obtained (Fig 6). The analysis of

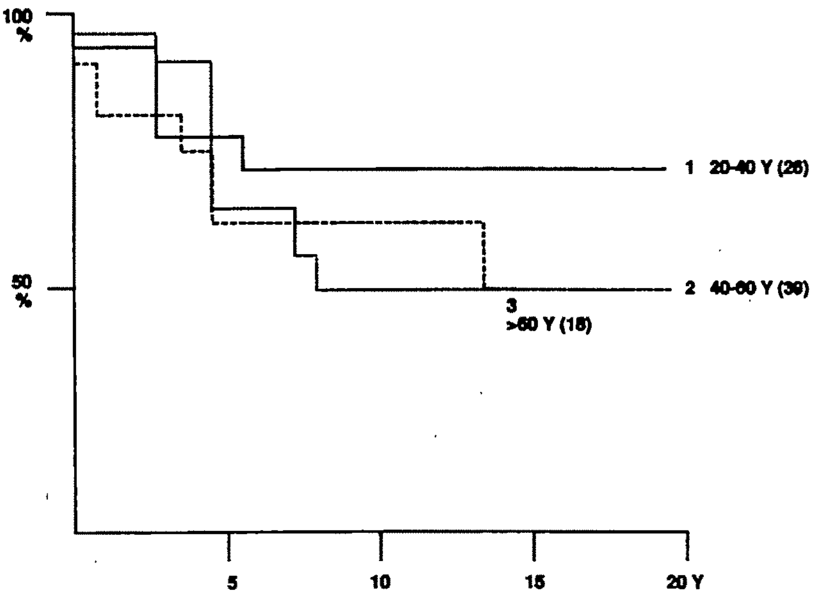

*Fig 2. Actuarial survival curves based on age of patients: 20 to 40 years, 40 through 60 years, and more than 60 years (overall, *$p = 0.94$*).*

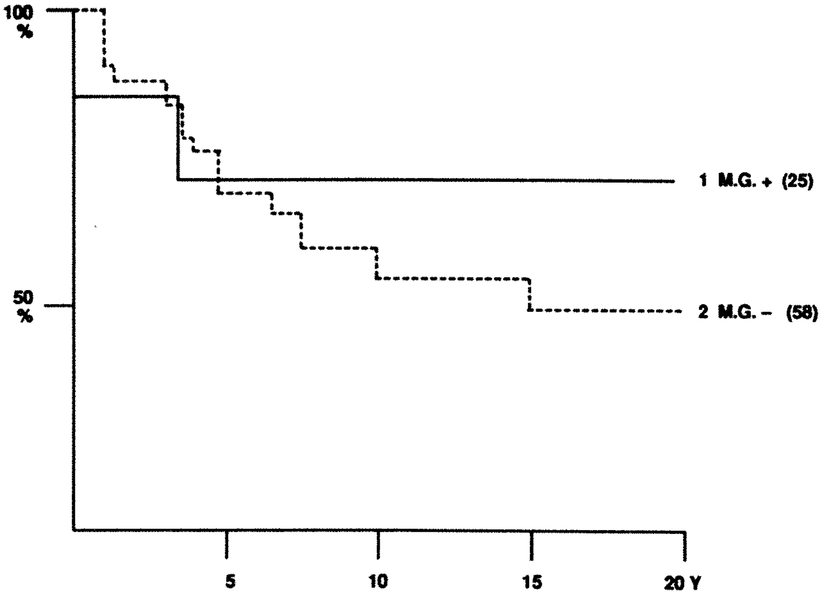

Fig 3. Actuarial survival curves for patients with and without myasthenia gravis (M.G.) (p = *0.82).*

this model proved to be highly significant ($p < 0.0001$). The multivariate analysis demonstrated that age ($p = 0.29$), sex ($p = 0.67$), association with MG ($p = 0.62$), and even histological type did not reach significance; the most significant prognostic factors were clinical stage ($p = 0.0004$) and clinicopathological staging ($p = 0.0001$).

Comment

The definition of prognostic factors is of paramount importance for the clinical and pathological staging of neoplasms and for the assessment of proper management. In this context, thymomas occupy a peculiar position because of their remarkable clinical and pathological polymorphism: they can occur as encapsulated, noninvasive, fully benign lesions or as largely invasive, even metastasizing tumors [1–8]. The wide spectrum of biological and clinical behavior of thymoma has led some authors [10, 16] to propose clinical staging procedures. These classifications, which proved to be of prognostic value, were based mainly on the degree of invasiveness of the tumor as assessed at operation and did not take into account any histological feature of thymoma. Conversely, attempts to correlate histology and prognosis have given rise to controversial conclusions [4–6].

In the present study, age, sex, and association with MG were not significant prognostic factors, but operative mortality was increased when thymoma was associated with MG, especially in elderly patients with stage III or IV tumors. Furthermore, patients with MG displayed a peak in mortality about 3 years postoperatively, which was partly an unsatisfactory response to medical treatment, whereas the death rate in patients without MG was more regularly distributed.

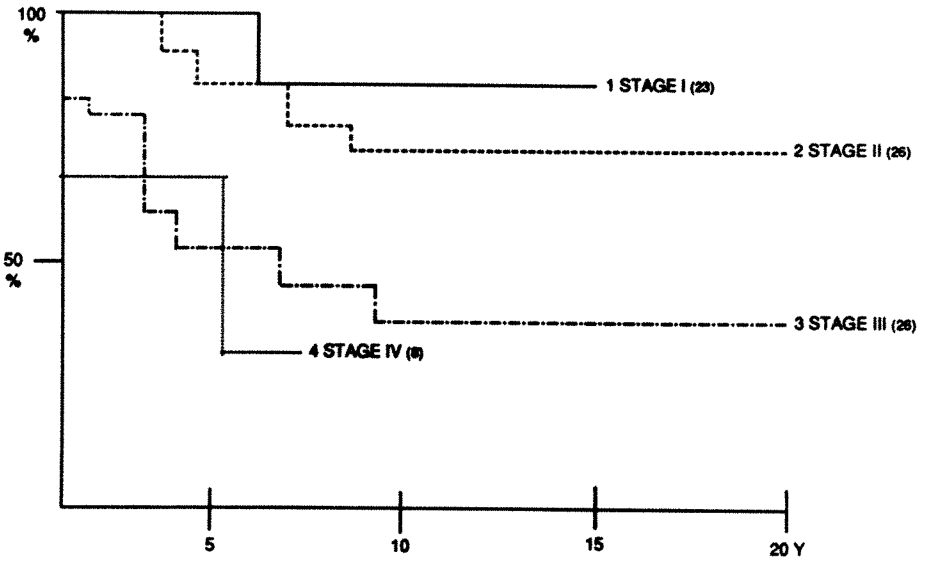

Fig 4. Actuarial survival curves based on clinical stage: 1 versus 2, p *= 0.30; 1 versus 3,* p < *0.01; 1 versus 4,* p < *0.01; 2 versus 3,* p < *0.01; 3 versus 4,* p = *0.69; and overall,* p < *0.001.*

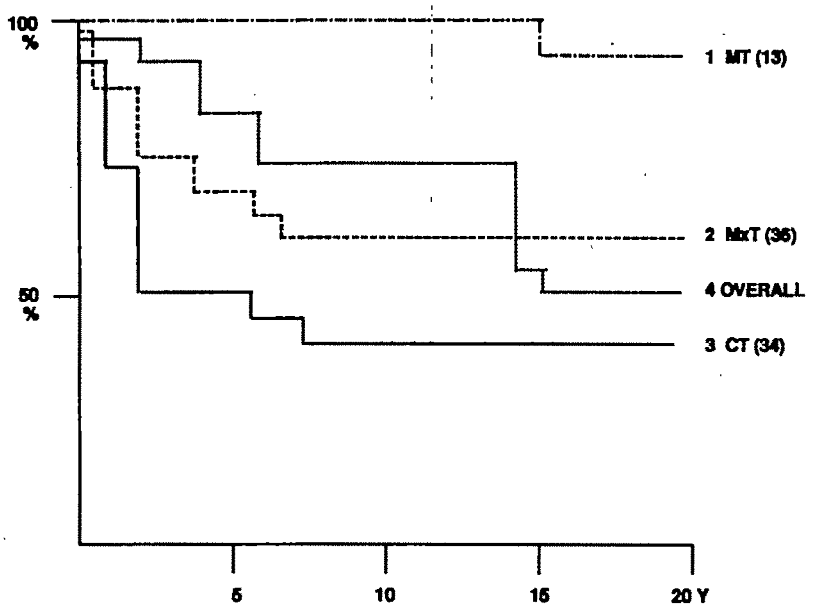

Fig 5. Actuarial survival curves based on histological type: 1 versus 2, $p < 0.05$*; 1 versus 3,* $p < 0.01$*; 2 versus 3,* $p = 0.12$*; and overall,* $p < 0.001$*. (CT = cortical type; MT = medullary type; MxT = mixed type.)*

The prognostic implication of associated MG in patients with thymoma is still controversial, however. Verley and Hollmann [17], Maggi and colleagues [6], and Lewis and co-workers [18] did not find a significant difference in the prognosis for patients with and those without MG. Conversely, Monden and co-authors [19] showed that thymoma without associated MG is more malignant than thymoma with MG. In the present study, the survival rate of patients with MG was higher than that of patients without MG, although the difference was not significant ($p = 0.82$). However, MG has always had an influence on outcome in terms of increased surgical risk.

We [7, 8] recently reported that the distinction of thymomas into cortical, medullary, and mixed types allows clinicopathological correlations and has a significant prognostic implication. This was confirmed in this study. In particular, tumors of the cortical type display a more aggressive behavior and are associated with a less favorable prognosis, with a peak in mortality within the first 3 years after surgical intervention; medullary-type tumors are, in most cases, fully benign; and tumors of mixed histology show intermediate behavior and have an intermediate prognosis.

The clinical stage is also a prominent prognostic factor. Stage I (noninvasive) and stage II (minimally invasive or moderately invasive) thymomas can be considered together as a group with a favorable prognosis; stage III (largely invasive) and stage IV (metastasizing) thymomas have a significantly worse prognosis. This finding may be of some clinical relevance, as a clear-cut distinction between stage I and stage II thymomas is not always feasible, whereas the distinction between stage II and stage III tumors is more obvious.

We propose a clinicopathological staging procedure

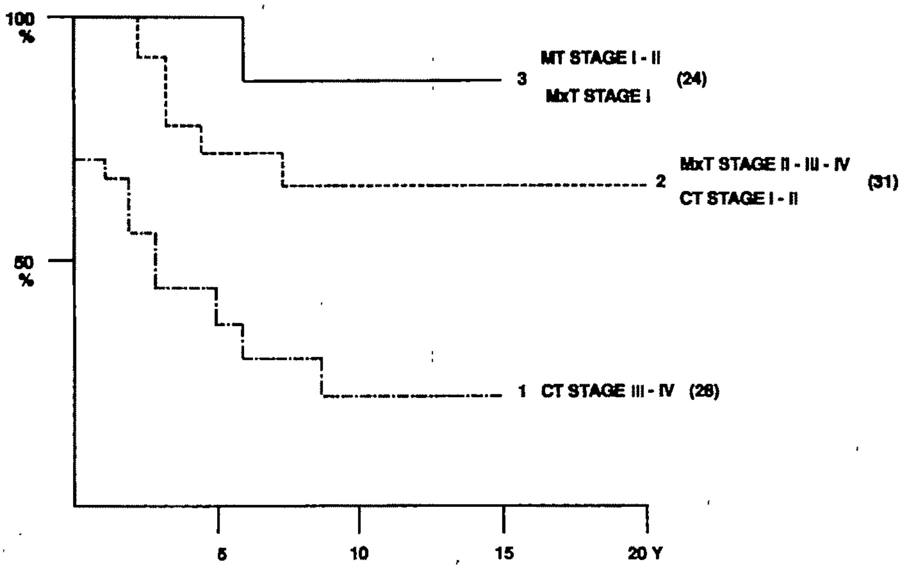

Fig 6. Actuarial survival curves based on clinicopathological stage: 1 versus 2, $p < 0.01$*; 1 versus 3,* $p < 0.01$*; 2 versus 3,* $p = 0.20$*; and overall,* $p < 0.0001$*. (CT = cortical type; MT = medullary type; MxT = mixed type.)*

based on both morphological (histological type) and clinical (clinical stage) grounds that is prognostically highly significant. The significance is higher for this model of clinicopathological staging ($p < 0.0001$) than for clinical stage ($p < 0.001$) and histological type ($p < 0.001$) considered individually, findings further supported by the multivariate analysis. This model may provide a prognostically highly significant approach to the problem of management of thymoma. Three major prognostic groups are included.

1. Stages I and II medullary type and stage I mixed type: This group has an excellent prognosis (survival, 100% at 5 years and 90% at 10 and 15 years). A radical operation with no postoperative therapy is the treatment of choice.
2. Stages I and II cortical type and stages II and III mixed type: This group has an intermediate prognosis (survival, 82% at 5 years and 75% at 10, 15, and 20 years). A radical operation is often possible, but postoperative radiotherapy is recommended for all patients. Stage IV mixed type is extremely rare (only 1 patient in our series). Postoperative radiotherapy and chemotherapy is suggested [20, 21].
3. Stages III and IV cortical type: This group has the least favorable prognosis (survival, 42% at 5 years and 27% at 10 and 15 years) with a higher incidence of operative and early mortality (within 3 years after operation). A radical operation should be attempted, and radiotherapy should always be administered postoperatively. Furthermore, chemotherapy should be prescribed for all stage IV patients and select stage III patients [20, 21].

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Intraoperative Ultrasonic Imaging of the Ascending Aorta in Ischemic Heart Disease

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In an attempt to locate any atherosclerotic lesion in the ascending aorta and to prevent embolization, intraoperative B-mode ultrasonography was performed in 100 patients with ischemic heart disease (31 women and 69 men). Ultrasonography was carried out with a 10-MHz probe placed directly on the ascending aorta. Ultrasonic imaging demonstrated an atherosclerotic lesion in the lower half of the aorta in 76 patients (76%), a lesion in the upper half of the aorta in 89 patients (89%), and a lesion at the orifice of the innominate artery in 99 patients (99%). Prospective palpation identified an atherosclerotic lesion in 12 (25%) of 48 patients. Thoracic computed tomography in 79 patients showed calcification in the lower half of the aorta in 6 patients (7.6%) and in the upper half of the aorta in 11 (13.9%). Palpation and thoracic computed tomography underestimated the frequency of atherosclerotic lesions. Intraoperative ultrasonography accurately identified atherosclerotic disease. This technique allows the surgeon to modify cannulation, aortic clamping, and operative technique to reduce the risk of perioperative stroke due to embolization of atherosclerotic debris from the ascending aorta.

(Ann Thorac Surg 1990;50:539–42)

A major cause of perioperative stroke is embolization of atherosclerotic material from the ascending aorta, which can result from insertion of an arterial cannula, aortic clamping, or establishment of the proximal anastomosis during coronary artery bypass grafting [1]. In an attempt to accurately determine whether atheromatous disease is present in the ascending aorta, intraoperative B-mode ultrasonic imaging of that structure has been performed.

Material and Methods

One hundred patients undergoing operation for ischemic heart disease were prospectively examined intraoperatively for atherosclerosis in the ascending aorta. The patients were seen from August 1987 to April 1989. The mean age of the patients was 62.9 years (range, 36 to 79 years), and 31 (31%) were women. The patients were consecutive for the most part. In a few instances, a sterilized ultrasonic probe had not been prepared. Ninety-nine patients underwent coronary artery bypass grafting, and 1 patient with familial hyperlipidemia of the homotype who had had coronary artery bypass grafting 2 years previously underwent aortic valve replacement with endarterectomy of the ascending aorta under the diagnosis of supraaortic and aortic stenosis because of progressive lipid deposition.

After median sternotomy and suspension of the heart in a pericardial cradle, visual and tactile examinations were performed. Ultrasonic imaging of the ascending aorta was then done using an Aloka SSD 125 system with a 10-MHz probe with a 4.2-cm sonolucent water bag. The probe was placed directly on the ascending aorta. The aorta was imaged in transverse and longitudinal views from the aortic annulus to the origin of the innominate artery (Fig 1). The scans were recorded on standard VHS videotape and displayed intraoperatively for assessment.

The results of the scans were compared with other findings. Based on the results of the ultrasonic imaging, modifications in the usual cannulation sites and proximal graft anastomotic sites were made by the surgeon as necessary.

Results

By ultrasonic imaging, 89 patients (89%) had an atherosclerotic lesion in the ascending aorta. Thoracic computed tomography was performed in 79 of the 100 patients and revealed calcification in the lower half of the aorta in 6 (7.6%) and in the upper half of the aorta in 11 (13.9%). Ultrasonic imaging in the same patients demonstrated an atherosclerotic lesion in the lower half of the aorta in 58 (73%) and in the upper half of the aorta in 66 (84%). By visual and digital examination of the aorta, 12 (25%) of 48 patients were found prospectively to have an abnormal aorta. Forty-two (87.5%) of these 48 patients had an abnormal aorta by ultrasonic imaging. Palpation and thoracic computed tomography both underestimated the frequency of an atherosclerotic lesion in the ascending aorta.

For reviewing the data, we divided the patients into two groups based on the results of ultrasonic imaging. Group 1 had minimal to mild atheromatous findings, thin intimal atheroma, and minimal to mild atheromatous

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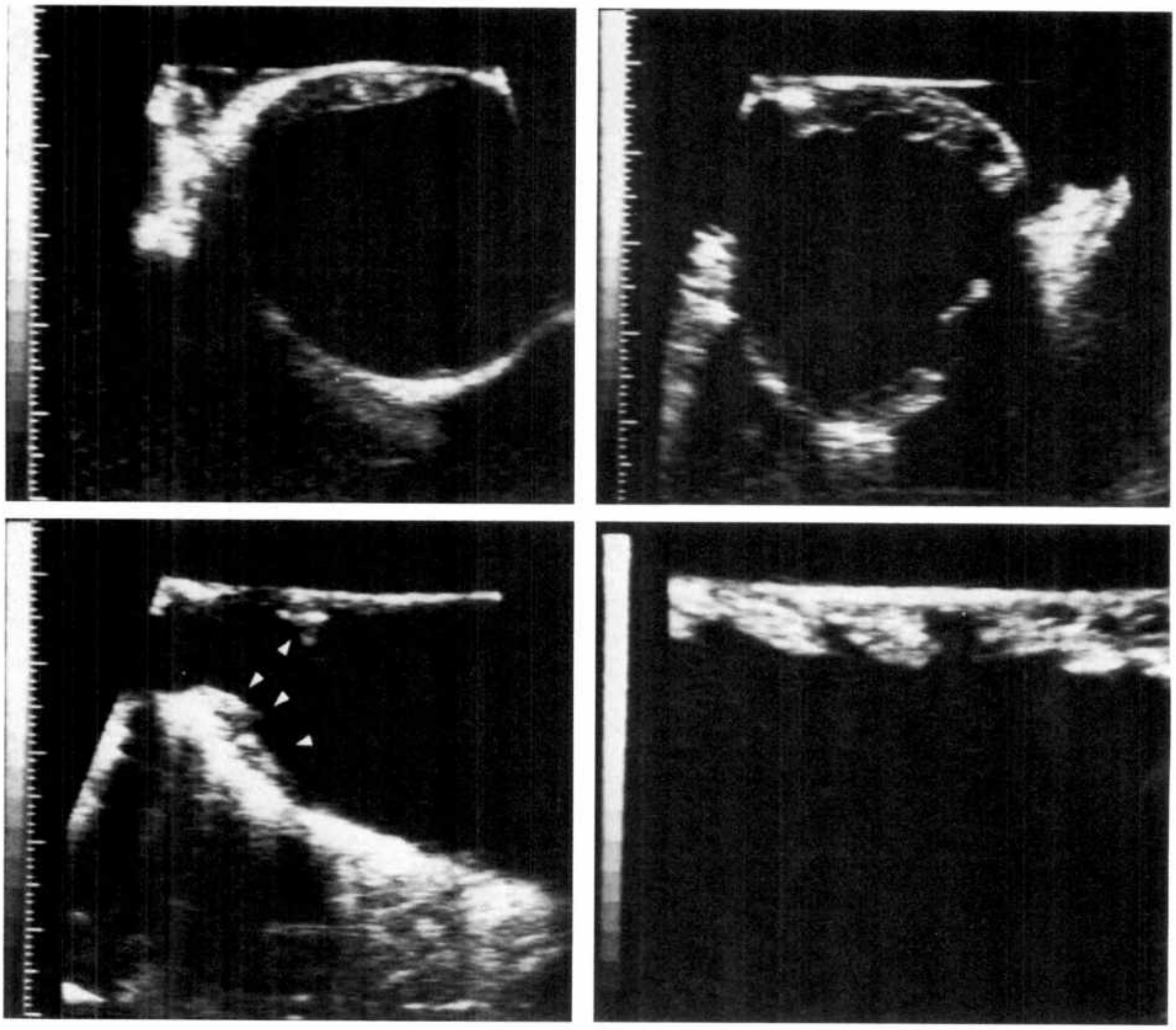

Fig 1. Intraoperative ultrasonic imaging. (Upper left) Transverse section of inferior ascending aorta; moderately hypertrophied anterior wall atheroma is seen. (Upper right) Transverse section of superior ascending aorta; severe atheromatous lesion with irregular intimal layer and focal ulceration is seen. (Lower left) Longitudinal section of ascending aorta; severe atheroma (white arrows) *is visible near the orifice of the innominate artery. (Lower right) Magnified view of a longitudinal section of the anterior wall of the ascending aorta; there is a moderate to severely hypertrophied intimal layer associated with multifocal ulceration. This patient has a minimal to moderate atherosclerotic lesion of the lower portion of the ascending aorta and a moderate to severe atheromatous lesion of the upper portion and at the orifice of the ascending aorta.*

ulceration with minimal calcification, and group 2 had moderate to severe atheromatous findings, thick intimal atheroma, and moderate to severe atheromatous ulceration with calcification.

The two groups were compared in terms of age, sex, and total cholesterol value. In group 1, the mean age was 59.2 ± 7.9 years (range, 39 to 78 years), there were 27 women and 11 men, and the mean total cholesterol value was 6.16 ± 0.83 mmol/L (238 ± 32 mg/dL). In group 2, the mean age was 64.3 ± 6.9 years (range, 40 to 79 years), there were 4 women and 58 men, and the mean total cholesterol value was 6.36 ± 0.93 mmol/L (246 ± 36 mg/dL). There were differences in frequency and severity of atherosclerosis of the ascending aorta between men and women, but there were no significant differences in age and total cholesterol value between groups 1 and 2.

Twelve patients in group 2 had a severe atheromatous condition warranting either a different cannulation site from the usual area in the ascending aorta or a different location for the proximal anastomosis. Three other patients in group 2 had cannulation of the femoral artery rather than the ascending aorta. Although recommended, the perfusion technique was not changed for 3 patients. Two of them had a moderate atherosclerotic obstructive lesion in both iliac arteries. The other patient was having emergency operation under cardiac massage.

There were two operative deaths, both in group 2: 1 patient died of multiple cerebral infarctions (the patient operated on emergently under cardiac massage) and 1, of low-output syndrome. Postoperative cerebral infarction occurred in 2 patients in group 2 and in none in group 1. One of these 2 patients had required a change in cannu-

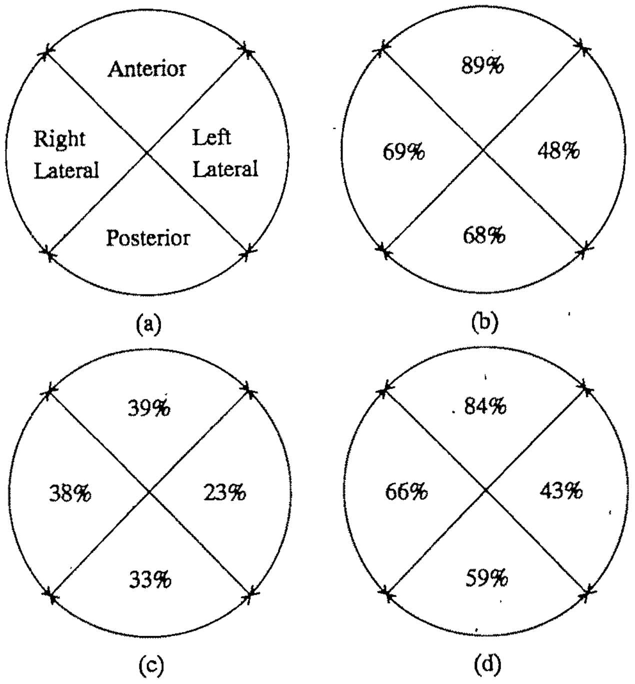

Fig 2. The incidence of atherosclerotic lesions in the ascending aorta: (a) Division of ascending aorta into four areas: anterior, right lateral, posterior, and left lateral. (b) Results in the entire ascending aorta, (c) lower half of the ascending aorta, and (d) upper half of the ascending aorta.

lation from the ascending aorta to the femoral artery. The other was 1 of the 12 patients who had received a change in the cannulation site in the ascending aorta.

For assessment of the incidence of atherosclerotic lesions, the circumference of the aorta was divided into four areas: anterior, right lateral, posterior, and left lateral (Fig 2). An atheroma was found in the anterior area in 89 of the 100 patients, in the right lateral area in 69, in the posterior area in 68, and in the left lateral area in 48. Atheromatous change was more prevalent in the upper half of the ascending aorta than in the lower half for both groups 1 and 2. The anterior–right lateral area of the ascending aorta had much more disease than the posterior–left lateral area both in the upper and lower halves of the ascending aorta.

Ninety-nine patients had a mild to severe atheromatous lesion near the orifice of the innominate artery. It is difficult to diagnose aortic arch lesions, including aortic dissection, by conventional methods, and direct ultrasonic imaging is necessary for improved accuracy in the diagnosis.

There were no wound infections in any of the patients studied.

Comment

Atheromatous emboli from the ascending aorta are usually related to either the placement of a cannula in the ascending aorta for cardiopulmonary bypass or the placement of a vascular clamp on the ascending aorta [1]. Complications relating to the diseased ascending aorta have been demonstrated to be a primary cause of unexpected death after coronary artery bypass grafting. In our hospital, we have found 25% of deaths after coronary artery bypass grafting to be related to perioperative stroke (unpublished data). Although calcification or any thickening in the outer layer of the ascending aorta can usually be determined by palpation, identification of soft, fragile atheroma in the aorta can be very difficult. Disease of the inner layer of the aorta may not be palpated, and palpation can dislodge atheromatous material into the ascending aorta.

Ultrasonic imaging eliminates the need for digital palpation of the aorta. Only an imaging probe, placed on the aorta without compressing the aorta, is necessary to obtain good imaging of the arterial wall. The technique is simple and rapid, adding only a few minutes to the operative procedure. Interpretation of the scan is easy with the visualization of the arterial wall. The 10-MHz Aloka probe provided excellent resolution of intraluminal detail because of its high frequency and short focus length compared with the conventional 7.5-MHz probe. Ultrasonic imaging in children with congenital heart disease or in adults with valvar disease showed almost normal or minimal findings in the ascending aorta with both the 10-MHz and 7.5-MHz probes. Consequently we believe the 10-MHz probe did not demonstrate oversensitive or false-positive findings.

In practice, any modification in operative technique involves relocation either of the arterial cannula in the ascending aorta or of the proximal anastomosis site for coronary artery bypass grafting. Relocation of the arterial cannula to the femoral artery was recommended in 3 patients but could not be performed because of bilateral iliac atherosclerotic obstructive lesions and the fact that cardiopulmonary bypass through the femoral artery was not reliable in 2 (with no apparent neurological deficit) and because of an emergency procedure under cardiac massage in 1 patient (who died of multiple cerebral infarctions postoperatively). One of the 12 patients who had required a minimal change in cannulation site demonstrated a mild neurological deficit postoperatively.

Numerous techniques have been introduced in an attempt to reduce the frequency of this complication. These include placing the proximal saphenous vein graft anastomosis on the internal mammary artery [2, 3], performing the cardiac procedure using hypothermic fibrillatory arrest to avoid clamps on the ascending aorta [4], doing ascending aortic endarterectomy with patch aortoplasty or ascending aortic replacement with hypothermic circulatory arrest without the use of vascular clamps [1], and creating the proximal anastomosis during coronary artery bypass grafting under aortic clamping, as we have routinely done in our hospital.

Tobler and Edwards [5] reported the frequency and location of atherosclerotic plaques in the ascending aorta in 100 autopsy specimens of coronary heart disease. They found little disease in the posterior area in the lower half of the aorta, a finding similar to our results, and substantial amounts in the posterior area in the upper half of the aorta, a finding in contrast to our results. There may be differences in definition between their specimens and our clinical cases. Marshall and colleagues [6] reported the anatomical locations of atherosclerotic plaques using intraoperative ultrasonic imaging. They found a very small incidence in the left lateral area, less than in our results, and a high incidence near the orifice of the innominate artery, similar to our results. This finding indicates that the arterial cannulation tube should be directed away from the innominate artery. The incidence of atherosclerotic lesions is likely related to age and cholesterol level, although not significantly so. There were large differences between men and women in frequency and severity of atherosclerotic lesions of the ascending aorta. This may or may not be specific to Japanese patients and should be further investigated.

In summary, intraoperative ultrasonic imaging identifies atherosclerotic disease in the ascending aorta. It is more accurate than digital palpation, and it allows the surgeon to modify the surgical procedure as required to minimize the risk of stroke due to atheroembolism. The technique is rapid and simple, and we have adopted it as a rule for cardiac procedures.

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Prevention of Paraplegia During Aortic Operations

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Ischemic spinal cord injury after aortic cross-clamping may be produced by a steal phenomenon. The present study investigates this possibility by directly measuring the oxygen tension on the spinal cord surface in pigs. After simple clamping of the aorta, oxygen tension decreased significantly distal to the clamping site both after occlusion of the thoracic aorta at T3-4 (group 1) and after occlusion of the abdominal aorta at L-1 (group 2). Exclusion of the thoracic aorta by a second clamp at T-13 restored oxygen tension almost to the original level, whereas segmentation of the abdominal aorta up to S-1 hardly affected oxygen tension in the area of the artery of Adamkiewicz in most of the animals. We conclude that after aortic cross-clamping, blood tends to drain away from the spinal cord rather than supplying it longitudinally. Without knowledge of the position of the Adamkiewicz artery in humans as well as of the competence of the collateral circulation in the excluded segment, it is necessary to develop a new strategy for repair of the aorta. We describe and discuss two surgical techniques for the prevention of paraplegia after aortic cross-clamping: the counterocclusion technique and the bypass fractionated technique.

(Ann Thorac Surg 1990;50:543–52)

The present understanding of the vascularization of the spinal cord is based on its first description in 1690 by Vieussens and on the anatomical studies of Adamkiewicz [1] and Kadyi [2]. According to Adamkiewicz and Kadyi, the anterior spinal artery is not an artery in the conventional sense but rather a heterogeneous anastomosed chain of numerous radicular arteries. This chain shows narrow areas at various heights with a minimal supply in the upper thoracic area. The number of radicular arteries making up this chain or perimedullar system is variable (less than five radicular arteries = paucisegmental and more than five = plurisegmental). The last and most important vessel of the spinal cord is the artery of Adamkiewicz or the arteria radicularis magna anterior (ARMA), which arises 75% of the time from the intercostal artery and 25% of the time from the lumbar artery as an arterial branch of the extramedullary system [3]. From this pattern of vascular supply to the spinal cord, Adamkiewicz developed the theory of partial or segmental flow. According to this theory, perfusion of the spinal cord occurs through partial flow in all branches of the radicular arteries. This theory would finally be confirmed in the 20th century through angiography [4].

In 1956, Adams and van Geertruyden [5] attempted to explain from the viewpoint of vascular morphology how the spinal cord is supplied with blood after the descending aorta (DA) has been clamped. They maintained that vascular perfusion of the spinal cord during the period the DA was clamped occurred proximally to distally by means of the anterior spinal artery. Through reduction in the caliber of this artery, as is true in the plurisegmental type, perfusion is critically reduced proximally to distally and results in spinal cord injury. Other authors [6, 7] believed that an effective perfusion pressure of 20 mm Hg in the DA distal to the clamp produces sufficient perfusion for the spinal cord to remain free from neurological consequences. Accordingly, they assumed that the spinal cord was supplied distal to the clamping of the aorta.

From vascular morphological and hemodynamic standpoints, we [8, 9] entertained qualified doubts about these views in our earlier studies. In our experiments, the clamping of the aorta resulted in a pressure gradient between the vessels perfusing the spinal cord and aorta distal to the clamping. From this we concluded that during the period of clamping, blood must flow back to the hypotonic aorta and evoke a steal phenomenon resulting in spinal cord injury.

The present study, therefore, addressed the changes in oxygen tension (Po_2) in the spinal cord after simple or segmental occlusion of the thoracic and abdominal aorta by directly measuring the Po_2 in the spinal cord to offer stronger, more direct evidence in support of the steal phenomenon. Possible surgical techniques to prevent spinal cord injury are also discussed.

Material and Methods

Twenty pigs weighing 25 to 30 kg were divided into two groups. The animals were intubated to receive nitrous oxide and oxygen (3:1) after premedication with 0.5 mg of atropine sulfate (Drobena), 250 mg of metomidate (Hyprodil), and 200 mg of azaperon (Stresnil). Metomidate and azaperon were given as needed during the operation. The animals were ventilated with a positive end-expiratory pressure of 6 mm Hg. Optimal oxygenation was assured by regular blood gas analyses. The

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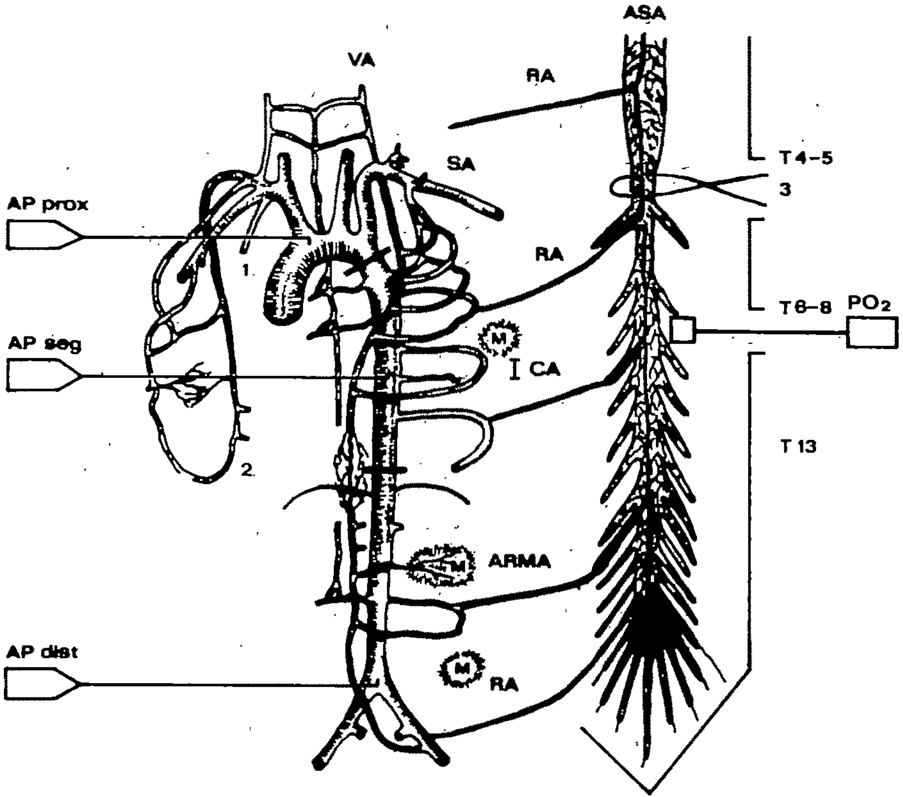


Fig 1. Experimental design in group 1. (AP dist = *distal arterial pressure [iliac artery];* AP prox = *proximal arterial pressure [aortic arch];* AP seg = *arterial pressure of descending aorta between T-4 and T-13;* ARMA = *arteria radicularis magna anterior [artery of Adamkiewicz];* ASA = *anterior spinal artery;* ICA = *intercostal artery;* M = *blood supply to body;* Po_2 = *oxygen tension measured on spinal cord surface between T-7 and T-8 before and after* 1 = *simple cross-clamping of descending aorta between T-3 and T-4;* 2 = *after double aortic cross-clamping at T-13; and* 3 = *after occlusion of spinal cord blood supply at T-5;* RA = *radicular artery;* SA = *subclavian artery;* VA = *vertebral artery.)*

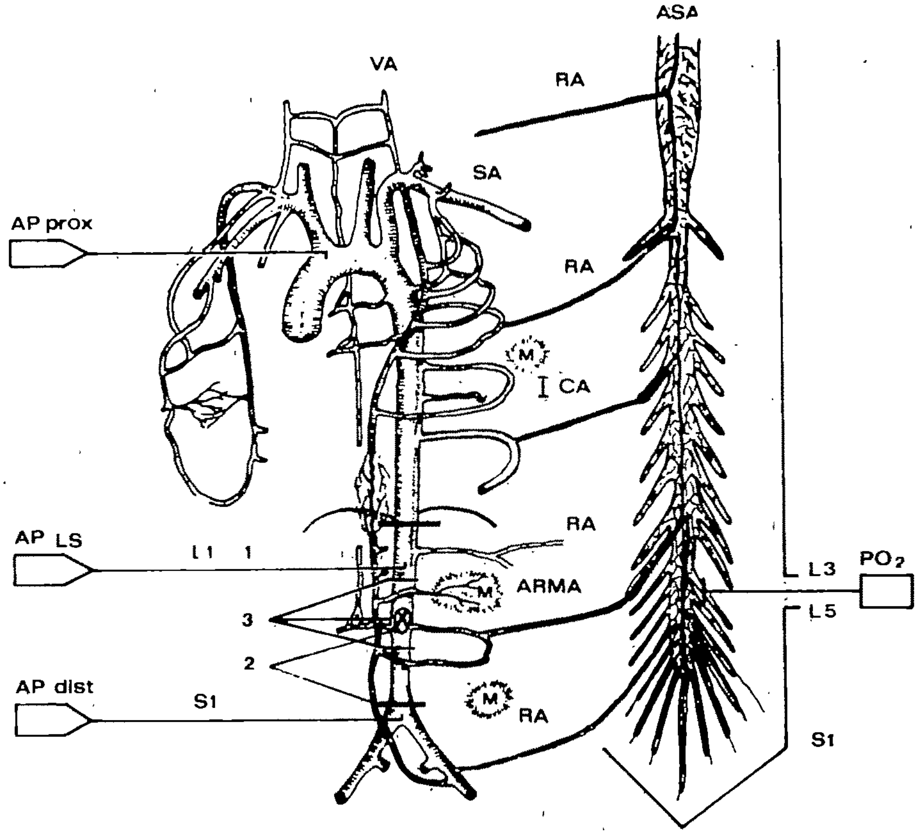


Fig 2. Experimental design in group 2. (AP_{LS} = *arterial pressure of lumbar aorta between L-1 and S-1;* Po_2 = *oxygen tension measured on spinal cord surface between L-3 and L-5 [area of arteria radicularis magna anterior or* ARMA*] before and after* 1 = *simple cross-clamping of abdominal aorta at L-1;* 2 = *after double cross-clamping of abdominal aorta at S-1, including clamping of anterior organ arteries; and* 3 = *after segmentation of abdominal aorta; other abbreviations are the same as in Figure 1.)*

Table 1. Measurements of Arterial Blood Pressure in Aortic Segments During Cross-Clamping of Descending Thoracic Aorta in Group 1

Animal No.	1–6 min	7–15 min		15–40 min		
	AP_{prox} (Aortic Arch) Before Clamping of DA (mm Hg)	AP_{prox} After Simple Clamping of DA at T3-4 (mm Hg)	AP_{dist} After Simple Clamping of DA at T3-4 (mm Hg)	AP_{prox} After Second Clamping of DA at T-13 (mm Hg)	AP_{dist} After Second Clamping of DA at T-13 (mm Hg)	AP_{seg} Between T-4 and T-13 (mm Hg)
1	90	150	19	155	12	55
2	103	125	20	115	15	55
3	78	114	18	116	11	30–35
4	108	150	19	130	15	50
5	80	125	18	155	13	62
6	75	122	20	124	16	48
7	80	120	18	127	13	60
8	70	110	19	135	14	40–45
9	88	117	18	132	13	55
10	75	126	16	130	10	56
11	90	150	19	155	14	60
Mean ± SEM	85.2 ± 3.6	128.1 ± 4.5	18.6 ± 0.34	134 ± 4.2	13.3 ± 0.5	52.0 ± 2.7

AP_{dist} = distal arterial pressure (iliac artery); AP_{prox} = proximal arterial pressure (aortic arch); AP_{seg} = arterial pressure of descending aorta between T-4 and T-13; DA = descending aorta; SEM = standard error of the mean.

proximal and distal aortic pressures were measured by appropriate cannulation. All animals received humane care in compliance with the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85–23, revised 1985).

Group 1

In group 1, after two small thoracotomies in the area of the fourth and 12th ribs, the DA was looped beyond the branching of the left subclavian artery at T3-4 and T-13 (Fig 1). A third arterial catheter was placed into the DA at T-7 for pressure measurement in this segment. Two laminectomies were performed at the levels of T-5 and T7-8. After the dura was opened, a dura thread ligature was placed at T-5. The body of the surface Po_2 electrode was fixed in a riding position on the surface of the spinal cord at T7-8 and was covered with warm compresses to prevent contact with the environment. The influence of

Table 2. Measurements of Oxygen Tension on Thoracic Spinal Cord (T-6 to T-8) During Cross-Clamping of Descending Thoracic Aorta in Group 1

Animal No.	1–6 min		7–12 min		15–30 min		30–40 min	
	Po_2 Before Clamping of DA (Baseline) (mm Hg)		Po_2 After Simple Clamping of DA at T3-4 (mm Hg)		Po_2 After Second Clamping of DA at T-13 (mm Hg)		Po_2 After Occlusion of Spinal Cord Blood Supply at T-5 (mm Hg)	
1	38.6	39.4	9.6	9.3	22.3	33.8	30.0	30.6
2	40.8	40.2	10.4	7.8	56.3	56.3	39.0	44.4
3	29.6	30.2	3.4	3.3	3.3	3.3	3.3	3.3
4	29.4	28.7	1.3	1.2	36.2	37.2	. . .	. . .
5	40.1	41.0	3.0	2.9	21.0	23.8	27.1	33.6
6	40.6	41.8	18.8	16.0	28.3	28.5	33.8	. . .
7	44.9	46.0	6.8	6.8	38.8	37.3	25.2	25.3
8	25.6	25.7	5.4	4.0	17.9	16.8	24.0	27.2
9	35.9	26.7	3.8	3.4	23.9	34.4	29.5	33.1
10	37.5	37.4	6.4	3.8	36.2	36.9	33.3	33.4
11	38.7	38.8	10.5	10.4	. . .	30.5	17.3	17.9
Mean ± SEM	36.5 ± 1.8	36.0 ± 1.9	7.2 ± 1.5	6.3 ± 1.3	28.4 ± 4.6	30.8 ± 4.0	26.3 ± 3.5	27.6 ± 4.0

DA = descending aorta; Po_2 = oxygen tension; SEM = standard error of the mean.

Table 3. Measurements of Arterial Blood Pressure in Aortic Segments During Cross-Clamping of Abdominal Aorta in Group 2

Animal No.	1–5 min		5–10 min		12–20 min	
	AP_{prox} Before Cross-Clamping of AA (mm Hg)	AP_{dist} Before Cross-Clamping of AA (mm Hg)	AP_{prox} After Simple Clamping of AA at L-1 (mm Hg)	AP_{dist} After Simple Clamping of AA at L-1 (mm Hg)	AP_{dist} After Second Clamping of AA at S-1 and Clamping of OA (mm Hg)	AP_{Ls} After Second Clamping of AA at S-1 and Clamping of OA (mm Hg)
1	80	75	120	15	13	22
2	88	80	124	14	12	24
3	75	70	120	16	14	28
4	74	70	110	18	16	35
5	76	75	100	11	10	20
6	85	80	105	17	16	29
7	77	73	108	13	13	43
8	83	79	112	15	14	25
9	82	73	109	16	12	45
Mean ± SEM	80.0 ± 4.5	75.0 ± 4.2	112.0 ± 7.2	15.0 ± 2.8	13.3 ± 2.6	30.1 ± 2.6

AA = abdominal aorta; AP_{dist} = distal arterial pressure (iliac artery); AP_{Ls} = arterial pressure of lumbar aorta between L-1 and S-1; AP_{prox} = proximal arterial pressure (aortic arch); OA = anterior organ arteries; SEM = standard error of the mean.

external oxygen diffusion from the air was effectively excluded, as superfusion of the electrode and tissue with pure oxygen did not change the Po_2 reading. Arterial blood pressures and Po_2 were measured before aortic cross-clamping, after simple aortic cross-clamping (T3-4), after double aortic cross-clamping (T3-4 and T-13), after occlusion of the spinal cord blood supply at T-5, and after restoration of aortic blood flow.

Group 2

In group 2, the abdominal aorta (AA) at L-1 and S-1 and the anterior organ arteries were looped through a left retroperitoneal approach (Fig 2). Blood pressure was measured in the AA between L-1 and S-1. A laminectomy was performed at L3-5 to measure the Po_2 in the area supplied by the ARMA. The Po_2 was measured before, after simple aortic cross-clamping (L-1), after double aortic cross-clamping (L-1 and S-1) including clamping of the anterior organ arteries, after segmentation of the AA between the lumbar arteries, and after restoration of aortic blood flow.

Measurements

The catheter and cannulas were connected to pressure transducers (Gould-Statham P23 ID; Gould Inc, Oxnard, CA), and all pressures were continuously monitored with

Table 4. Measurements of Oxygen Tension on Lumbar Spinal Cord (L-3 to L-5) in Area of Adamkiewicz Artery During Cross-Clamping of Abdominal Aorta in Group 2

Animal No.	2–5 min		7–12 min			15–20 min		25–35 min		
	Po_2 Before Clamping of AA		Po_2 After Simple Clamping of AA at L-1			Po_2 After Second Clamping of AA at S-1 including OA		Po_2 After Release of Clamps Until Perfusion of ARMA		
1	37.4	38.0	18.8	8.0	0.6	1.6	1.6	51.9	56.9	55.4
2	44.9	44.3	16.0	7.9	2.8	2.8	2.4	37.7	37.8	42.2
3	31.8	30.4	8.2	4.1	1.7	2.7	2.7	19.1	37.8	34.1
4	34.7	33.3	8.2	7.6	7.6	7.6	7.3	33.8	33.8	28.3
5	35.9	38.5	2.6	1.1	0.8	0.8	1.8	36.7	40.0	32.1
6	41.5	39.8	5.3	3.8	3.5	3.5	3.3	28.7	31.2	48.7
7	39.4	40.1	11.4	10.2	13.1	13.5	27.5	38.3	38.5	37.7
8	37.8	34.3	9.7	6.4	4.2	4.2	4.2	36.5	40.8	39.5
9	42.3	42.2	3.6	3.4	8.9	18.9	34.8	35.0	41.1	38.5
Mean ± SEM	38.4 ± 1.4	37.9 ± 1.5	9.3 ± 1.8	5.8 ± 1.0	4.8 ± 1.4	6.2 ± 2.4	9.5 ± 2.5	35.3 ± 2.9	39.8 ± 2.4	39.6 ± 2.9

AA = abdominal aorta; ARMA = arteria radicularis magna anterior (artery of Adamkiewicz); OA = anterior organ arteries; Po_2 = oxygen tension; SEM = standard error of the mean.

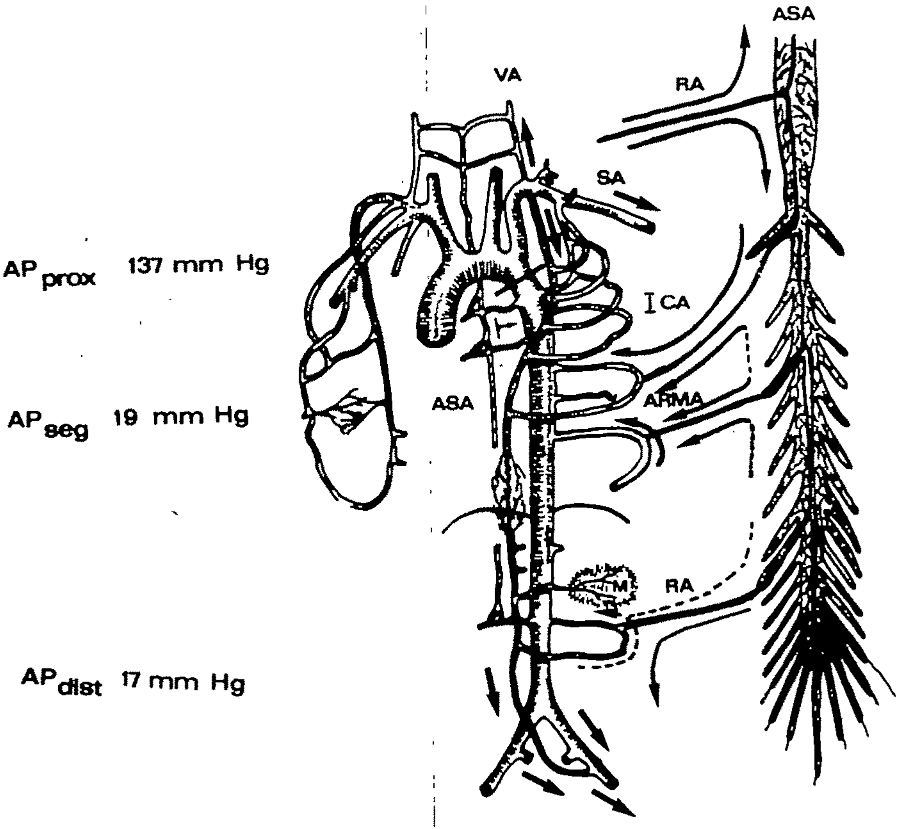


Fig 3. Mean pressure values and direction of blood flow after simple cross-clamping of descending thoracic aorta at T3-4 (steal phenomenon). Abbreviations are the same as in Figure 1.

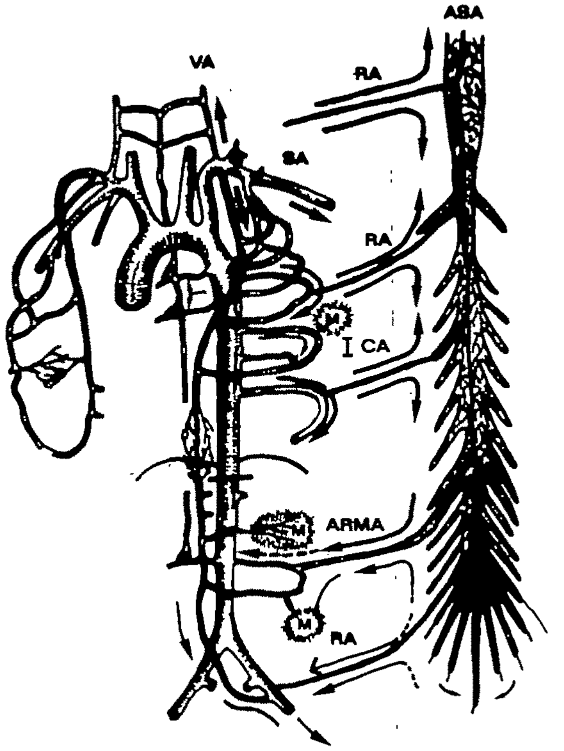


Fig 4. Cross-clamping of abdominal aorta at L-1 resulted in a steal phenomenon and spinal cord injury because the arteria radicularis magna anterior (ARMA) *lies distal to the clamping. Other abbreviations are the same as in Figure 1.*

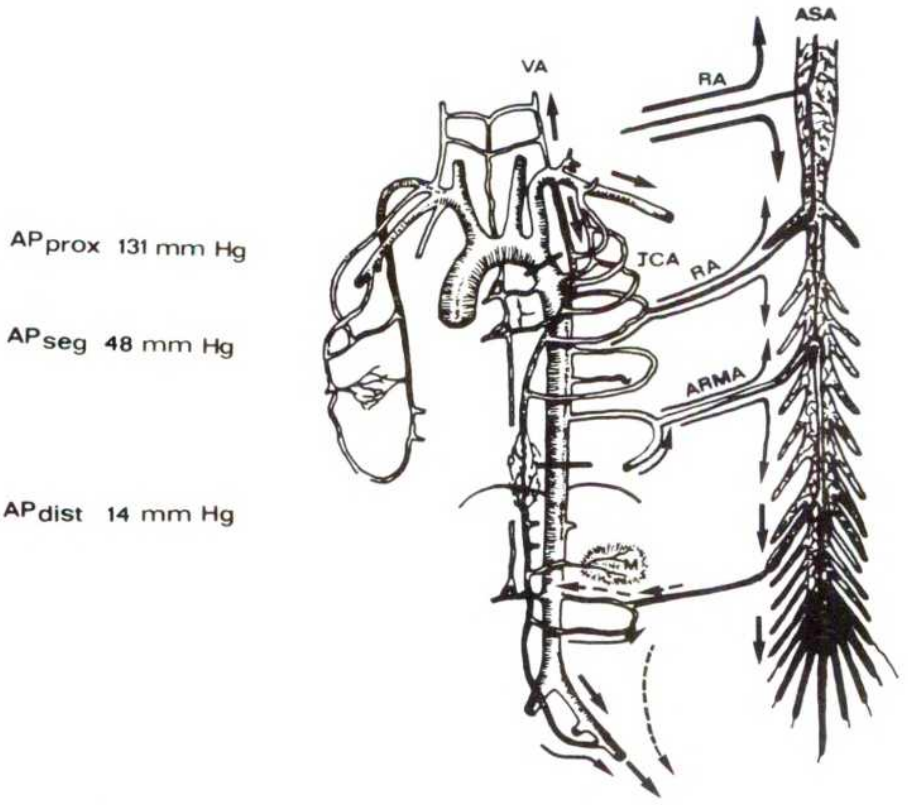
a six-channel recorder (Hellige & Co, GmbH, Freiburg, FRG). All pressure recordings in this presentation are expressed as electrically integrated means. The surface Po_2 measurements were performed with Hysol (Dexter Corp, New York, NY)-embedded and isolated gold cathodes (15 μm in diameter) arranged concentrically around a silver/silver chloride reference wire (200 μm in diameter), as described by Lübbers and associates [10]. The Po_2 electrode was covered with a thin (25-μm) polypropylene membrane and had a temperature sensor for continuous temperature measurement (Radiometer AG, Copenhagen, Denmark). The polarization tension was −800 mV. The Po_2 measurement was made on a six-channel compensation recorder (Linseis Co, Selb, FRG). Before and immediately after each experiment, the surface Po_2 electrodes were calibrated in 0.9% sodium chloride solutions at 36.5°C equilibrated with a gas mixture of known content (2% and 5% oxygen in nitrogen and purified nitrogen).

Body temperature was measured continuously with a rectal thermometer during operation and was kept relatively constant between 34.5° and 36.5°C with a heating lamp. The electrocardiogram was recorded continuously.

Statistical Analysis

For the statistical analysis of the results, only those data that showed deviations of less than 10% between the beginning and the end of the experiment were considered. Only those experiments were evaluated in which at

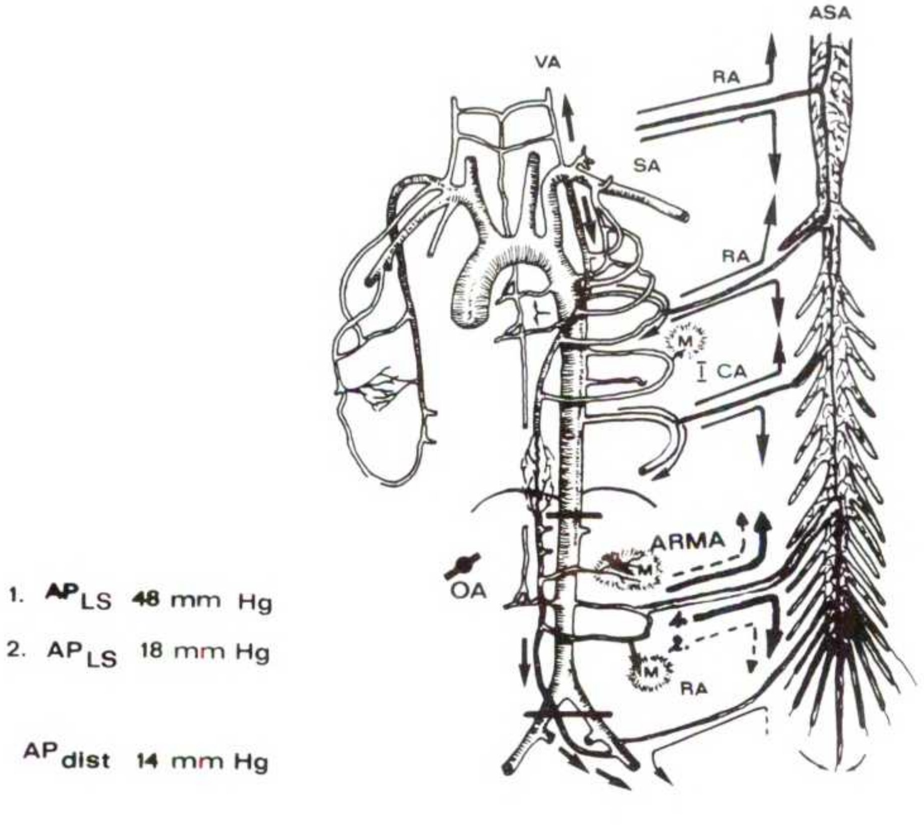
Fig 5. Mean pressure values and direction of spinal cord blood flow after second aortic cross-clamping at T-13. Segmental spinal cord blood flow proximally and between clamps. Prevention of steal phenomenon by second clamp distal to arteria radicularis magna anterior (ARMA). *Abbreviations are the same as in Figure 1.*



least three measurements of the electrode could be obtained without disturbance. The vascular morphology of the spinal cord was examined by postoperative anatomical dissection.

For the descriptive data analysis, contingency table analyses with unpaired and paired *t* tests were used. All computations were performed with the Statistical Package for the Social Sciences (CDC version 7.0) [11]. These

Fig 6. Mean pressure values and direction of spinal cord blood flow after cross-clamping of abdominal aorta at L-1 and S-1 including the anterior organ arteries (OA). *Prevention of steal phenomenon.* (AP_{LS} = *arterial pressure of the lumbar aorta between L-1 and S-1; 1. = no spinal cord injury: arterial pressure in excluded aortic segment is higher than critical closing level; 2. = arterial pressure in excluded aortic segment is lower than critical closing level; the arteria radicularis magna anterior [ARMA] should be promptly anastomosed to the graft [fractionated procedure]; other abbreviations are the same as in Figure 1.)*



programs were employed in the present study to maintain continuity and allow comparisons with our previous experiments.

Results

Group 1

Simple clamping of the DA at T3-4 resulted in a significant increase in arterial blood pressure proximal to the clamp to 150.3% of the original value (average change, 85.2 mm Hg to 128.1 mm Hg; $p < 0.001$) (Table 1). The blood pressure distal to the clamp decreased significantly to 21.9% of the original value (from an average of 85.2 to 18.6 mm Hg; $p < 0.001$).

Placing a second clamp on the DA at T-13 led to a significant second increase in the blood pressure of the excluded aortic segment T-4 to T-13 from 18.6 to 52.0 mm Hg (from 21.9% to 61.1% of the original value) ($p < 0.001$). The blood pressure distal to T-13 continued to decrease significantly from 18.6 to 13.3 mm Hg ($p < 0.01$).

After simple clamping of the DA at T3-4, the Po_2 fell significantly in the thoracic cord from 36.0 to 6.3 mm Hg ($p < 0.001$) (Table 2). After a second clamp was put on the DA at T-13, the Po_2 rose to 30.8 mm Hg on average ($p < 0.001$).

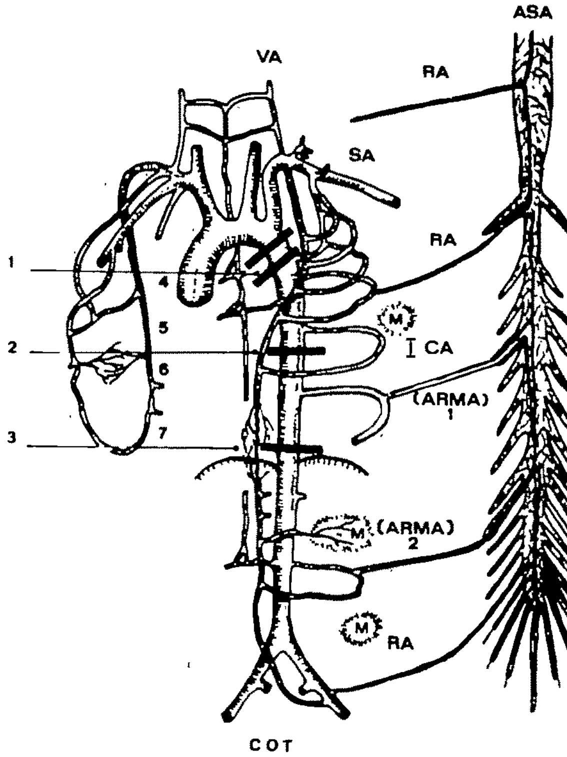


Fig 7. Counterocclusion technique (COT). (1 = proximal double aortic cross-clamping; 2 = distal aortic cross-clamping; 3 = counterocclusion of descending aorta at T-12 or distal to arteria radicularis magna anterior [ARMA]; 4 = proximal anastomosis; 5 = proximal clamp replaced distally; 6 = reimplantation of intercostal artery [ICA] and perfusion of ICA; 7 = distal anastomosis; other abbreviations are the same as in Figure 1.)

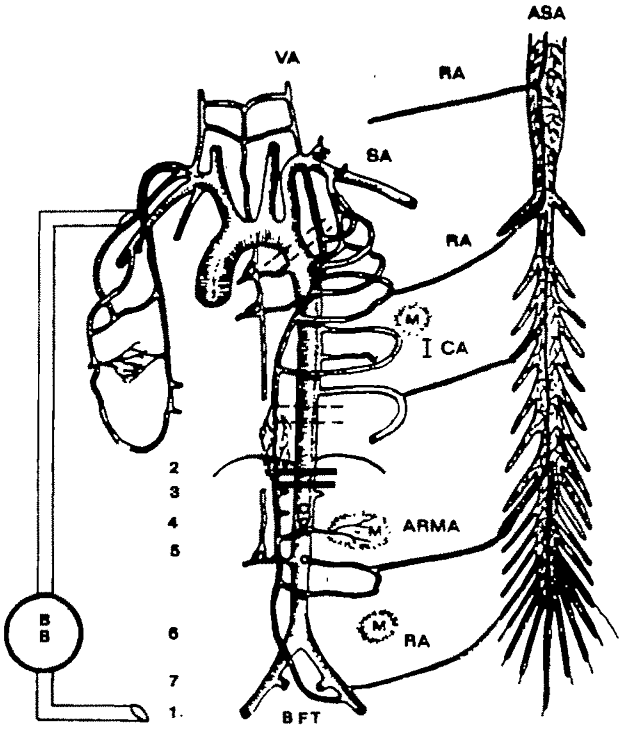


Fig 8. Bypass fractionated technique (BFT) for replacement of descending thoracic, thoracoabdominal, or abdominal aorta. (1 = bypass procedure [B]; 2 = proximal double cross-clamping of descending aorta; 3 = proximal anastomosis; 4 = reimplantation of intercostal [ICA] or lumbar artery [LA] [arteria radicularis magna anterior or ARMA] and reperfusion of ICA or LA [ARMA]; 5 = Reimplantation of visceral arteries and reperfusion of these arteries; 6 = distal anastomosis; 7 = end of bypass procedure; other abbreviations are the same as in Figure 1.)

The recurrent increase in the Po_2 in 10 of 11 animals correlates with the recurrent increase in the blood pressure of the aortic segment that was excluded; however, the increase had to be at least 35 to 40 mm Hg. During the period of double clamping of the DA when a proximal ligature of the spinal cord was placed at T-5 according to standardized procedures, no significant alteration of the Po_2 in the spinal cord at T7-8 resulted.

Group 2

After the clamping of the AA at L-1, the proximal arterial blood pressure increased significantly from 80.0 to 112.0 mm Hg (140% of the original value) (Table 3). Distal to the clamp, the arterial blood pressure decreased significantly from 75.0 to 15.0 mm Hg (20% of the original value). After the second clamping of the AA at S-1, including the anterior visceral arteries, the blood pressure increased significantly in the aortic segment L-1 to S-1 to 30.1 mm Hg ($p < 0.001$). The distal arterial blood pressure decreased an additional 2 mm Hg to about 13 mm Hg.

Simple clamping of the AA at L-1 proximal to the Adamkiewicz artery resulted in a significant average decrease in the Po_2 at L3-5 (supply area of the Adamkiewicz artery) from 37.9 to 4.8 mm Hg ($p < 0.001$) (Table

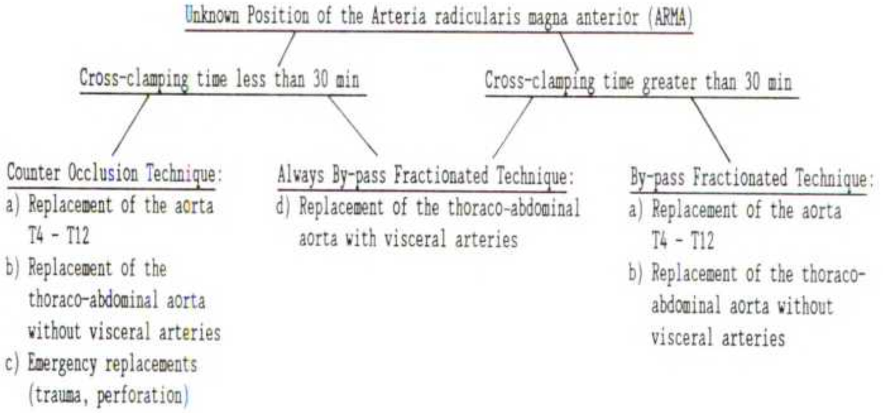

Unknown Position of the Arteria radicularis magna anterior (ARMA)
Cross-clamping time less than 30 min
Cross-clamping time greater than 30 min
Counter Occlusion Technique:
a) Replacement of the aorta T4 - T12
b) Replacement of the thoraco-abdominal aorta without visceral arteries
c) Emergency replacements (trauma, perforation)
Always By-pass Fractionated Technique:
d) Replacement of the thoraco-abdominal aorta with visceral arteries
By-pass Fractionated Technique:
a) Replacement of the aorta T4 - T12
b) Replacement of the thoraco-abdominal aorta without visceral arteries

Fig 9. Counterocclusion technique and bypass fractionated technique: recommended surgical techniques for replacement of descending aorta. Choice of technique when position of artery of Adamkiewicz is unknown and when cross-clamp time is less than or greater than 30 minutes.

4). Placing a second clamp on the AA at S-1, including the ventral visceral arteries, did not significantly influence the Po_2 in 7 of the 9 animals. The recurrent increase in the Po_2 in 2 of the animals correlates with the recurrent increase in the blood pressure of the aortic segment between L-1 and S-1; however, the increase in pressure had to be at least 35 to 40 mm Hg. After the release of the clamp, proximally to distally, until the Adamkiewicz artery was perfused from the aortic lumen, the Po_2 increased in the lumbar cord of all animals to more than the original value of 39.8 mm Hg ($p < 0.001$).

Comment

Spinal cord injury after aortic cross-clamping can occur independently of aortic disease in 3% to 33% of cases [8, 9]. Injury to the cord can also occur after various surgical procedures not involving cross-clamping of the DA and can develop spontaneously in association with dissection of the aorta or cardiac insufficiency [12]. Attempts to prevent this complication have included cooling and various methods of temporary or permanent bypass, but neurological complications were not prevented [12–14]. The answer to how this complication may be prevented lies in the resolution of how it arises.

The mode of development of paraplegia after clamping of the DA should be examined on the basis of the alteration in the Po_2 in the spinal cord and the changes in blood pressure in various aortic segments. After the DA was clamped at T3-4 and at L-1, not only did the Po_2 decrease in the spinal cord but also the blood pressure in the aorta distal to the clamp decreased massively (Figs 3, and 4; see Tables 1–4). The decrease in the Po_2 was in no way dependent on the variation in the anterior anastomosed chain. The chain is identical in all pigs and in 55% of humans (plurisegmental) [3, 15].

By directly measuring the Po_2, the possible influence of the spinal fluid pressure was eliminated, which is in contrast to other methods. The relative perfusion pressure between the aorta and the spinal cavity of 20 mm Hg could not compensate for the massive decline in the Po_2 in any animal. This finding refutes the assertion that a relative perfusion pressure between the aorta and the spinal column of 20 mm Hg guarantees a sufficient minimal supply without neurological consequences [6, 7]. In our previous studies [8, 12], spinal fluid drainage offered no protection against spinal cord damage.

These observations clearly show that during aortic cross-clamping, the spinal cord cannot be perfused from the hypotensive aorta as others [6, 7] have claimed. Instead, proximal clamping of the aorta must result in a steal phenomenon that tends to aggravate spinal ischemia. The placement of a second clamp at T-13 in group 1 and at S-1 (including clamping the organ arteries) in

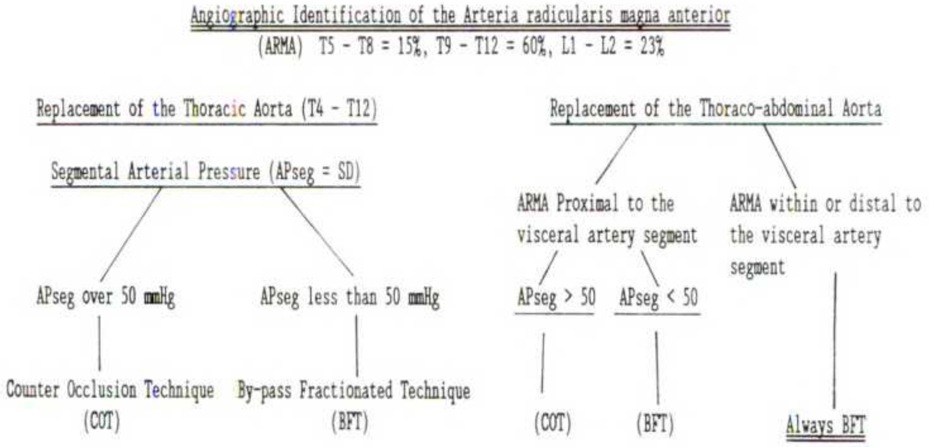

Angiographic Identification of the Arteria radicularis magna anterior
(ARMA) T5 - T8 = 15%, T9 - T12 = 60%, L1 - L2 = 23%
Replacement of the Thoracic Aorta (T4 - T12)
Replacement of the Thoraco-abdominal Aorta
Segmental Arterial Pressure (APseg = SD)
ARMA Proximal to the visceral artery segment
ARMA within or distal to the visceral artery segment
APseg over 50 mmHg
APseg less than 50 mmHg
APseg > 50
APseg < 50
Counter Occlusion Technique (COT)
By-pass Fractionated Technique (BFT)
(COT)
(BFT)
Always BFT

Fig 10. Counterocclusion technique and bypass fractionated technique: choice of technique based on segmental arterial pressure (APseg) and location of arteria radicularis magna anterior (artery of Adamkiewicz).

group 2 resulted in a rise of Po_2 to almost the original values in 10 of the 11 pigs in group 1 and in 2 of the 9 pigs in group 2 (Figs 5, 6). This can only mean that a steal phenomenon was effectively prevented by the second clamp. Consequently, double clamping seemed to have a beneficial effect on collateral circulation in most of the animals in group 1 and in a few in group 2. This was described in our previous hemodynamic study [9] and subsequently confirmed in humans (DA Cooley: personal communication).

The failure of ligation of the proximal cord (and therefore the anterior spinal artery) to ablate this response confirms that the main supply to the cord in pigs is through extramedullary collaterals with connections to the aorta and is not dependent on a contiguous anterior spinal artery as other authors assumed [5].

In contrast to Adams and van Geertruyden [5], it must be assumed that the perfusion proximal to the clamping and in the excluded segment is most likely segmental. A rise in the Po_2 of the spinal cord occurs only when the arterial pressure between T-4 and T-13 and the arterial pressure between L-1 and S-1 reach a minimum pressure of 40 mm Hg or when the ARMA is sufficiently perfused. Paraplegia results when the collateral circulation in the excluded region is not adequately developed or when the ARMA supplies the spinal cord distal to the clamp. Laschinger, Cunningham, and associates [16, 17] reported that a minimal pressure of 60 mm Hg distal to the clamp is necessary to prevent spinal cord injury. Our measurements show the importance of arterial blood pressure in the excluded aortic segment for spinal cord blood supply.

Proximal aortic cross-clamping in cases of acute traumatic aortic rupture is comparable with the situation created in our group 1 animals. In contrast to patients with chronic coarctation, this model has no developed collateral pathway, and the spinal cord circulation actually drains retrograde into the hypotensive aorta. This may explain the short permissible aortic clamping time in traumatic disruption demonstrated by Katz and coworkers [13]. On the other hand, the collateral circulation in coarctation of the aorta is usually well developed so that proximal clamping does not produce a substantial pressure drop in the distal aorta. Only in isolated instances is the collateral circulation so poorly developed that the spinal arterial pressure could fall below the critical closing level.

According to the present study and our previous study, paraplegia can be expected despite the use of a bypass procedure and despite adequate perfusion distal to the cross-clamp when the perfusion pressure in the excluded segment is not sufficient.

In consideration of the Po_2 in the spinal cord as well as the arterial pressure between T-4 and T-13 and between L-1 and S-1, the collateral circulation appears to be better developed in the thoracic region than in the lumbar region. This possibly explains the clinical experience that the rate of paraplegia increases after cross-clamping the DA proximally to distally [14, 18]. Furthermore, these animal experiments explain why the ligation of the lumbar arteries results in a higher rate of paraplegia than the ligation of the intercostal arteries [12, 19, 20].

Based on present knowledge, new strategies or additional strategies must be developed to minimize or even prevent spinal cord injury after cross-clamping of the DA. With reference to the results in the present study, these strategies must take into consideration several factors.

1. The presence of a steal phenomenon when the ARMA perfuses the spinal cord distal to the cross-clamp.
2. The exact position of the ARMA in relation to the cross-clamp (angiographic determination of the ARMA) [21].
3. The condition of the collateral circulation, ie, measurement of the segmental arterial pressure between T-4 and T-13 or between L-1 and S-1, possibly by means of a cross-clamp.
4. No prolongation of the 20-minute ischemia tolerance time of the spinal cord [22]. Here it is necessary to differentiate between the occlusion time of the aorta and the duration of ischemia of the spinal cord.
5. The avoidance of a permanent ligature of radicular arteries, such as in the ARMA. This is necessary for the perfusion of the spinal cord [9, 12, 23].

Two techniques that might greatly reduce the rate of paraplegia or even prevent this complication are the counterocclusion technique and the bypass fractionated technique (Figs 7–10). The counterocclusion technique includes the placement of an additional clamp distal to the ARMA or at T-12 for repair of the aorta after damage or rupture. The bypass fractionated technique requires the use of a sufficient bypass procedure, sufficient meaning a minimal perfusion pressure of 50 mm Hg and a minimal perfusion volume of 2 L/min. The aorta is cross-clamped in stages proximally to distally. After each surgical step, the proximal clamp is replaced distally and blood flow is released proximally. With this procedure, the time of ischemia of the spinal cord is much shorter than the occlusion time of the aorta and equals the time necessary to perform one anastomosis. The angiographic determination of the position of the ARMA and the measurement of the segmental pressure are valuable in choosing the appropriate technique for replacement of the aortic segments.

Laschinger [23], Maeda [24], and their associates assessed the adequacy of spinal cord blood flow during aortic occlusion and located important intercostal arteries critical to spinal cord blood flow with somatosensory evoked potentials. Maeda and colleagues concluded that the maintenance of relative spinal cord perfusion pressure at more than 40 mm Hg by increasing mean distal aortic pressure, withdrawing cerebrospinal fluid, or both is an effective procedure for preventing postoperative paraplegia.

In contrast to Laschinger and co-workers [23], Crawford and colleagues [14] found that somatosensory evoked potentials had no major impact on the prevention of neurological deficits during or after replacement of the DA. We and others [8, 12, 25] found that spinal fluid drainage had no substantive effect on spinal cord injury.

Furthermore, we believe that the bypass fractionated technique will make the use of somatosensory evoked potentials superfluous.

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Combined Internal Mammary Artery Graft for Coronary Artery Revascularization

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Five patients with multiple-vessel coronary artery disease underwent isolated coronary artery bypass grafting with a technique involving both internal mammary arteries and a small piece of interposed saphenous vein. The combined internal mammary artery grafts were used for sequential grafting. A total of 20 anastomoses were performed (average number, 4 anastomoses per patient). There were no operative deaths. Postoperative complications included reoperation for bleeding in 1 patient and diaphragmatic dysfunction in another. Postoperative coronary angiography 2 days before discharge (mean time, 10 days postoperatively) revealed that all the sequential anastomoses with the combined IMA graft were patent. Exercise tolerance tests performed 3 and 11 months postoperatively indicated excellent results and no ischemia. Based on this experience, we conclude that this method appears promising for multivessel coronary artery bypass grafting.

(Ann Thorac Surg 1990;50:553–6)

Limitations of the traditional saphenous vein coronary bypass operation center on the marked susceptibility of this conduit to degeneration and atherosclerosis with reduced flow and early return of symptoms. Several studies [1, 2] noted this susceptibility both early and late postoperatively. Patients receiving internal mammary artery (IMA) grafts, however, have been reported to have significantly improved survival [1, 3]. The results with bilateral and sequential IMA grafts late postoperatively have led to a new era in coronary artery surgery [3–5]. Using the IMA to bypass more coronary arteries may be an important means of improving longevity after coronary artery revascularization [4, 6, 7]. The main goal in using the left (LIMA) and right (RIMA) IMAs combined with the interposition of a small piece of saphenous vein is to expand the use of an ideal conduit to as many situations as possible and to reach areas of myocardium that could not previously be served by the IMA [6, 8].

In this study, we present our limited experience with 5 patients who underwent coronary artery bypass grafting with a technique that permits revascularization of the entire myocardium with the IMAs.

Material and Methods

Patient Population

In the 3-month period March through May 1989, 5 male patients with multiple-vessel coronary artery disease underwent isolated coronary artery bypass grafting at the Institute of Cardiology, University of Istanbul, with our technique. The patients ranged in age from 50 through 63 years old.

Preoperative symptoms were defined by the Canadian Heart Association classification. There was only 1 patient in class 2; the rest were in class 3. Preoperative coronary angiography revealed triple-vessel disease in 4 patients and double-vessel disease in 1 patient. One patient had total occlusion of the left main coronary artery and another, clinically significant left main stenosis.

All patients had good left ventricular function, and no patient had sustained a myocardial infarction. Clinical data are shown in Table 1.

Technique of Operation

A median sternotomy is made. Both the RIMA and LIMA are dissected from the sternum with electrocautery in the usual fashion, starting from the right side. The arteries are mobilized with the pedicle and divided distally just before the superficial epigastric and musculophrenic bifurcation. The decision to use a combined IMA graft is made only if the flow in the artery is more than 100 mL/min and the diameter is at least 1.5 mm. The RIMA is prepared as a free graft by cutting proximally at the junction between it and the subclavian artery. A piece of saphenous vein 3 to 4 mm in diameter is first anastomosed to the distal end of the LIMA with 7-0 Prolene (Ethicon, Somerville, NJ) and continuous suture technique, resulting in a fish-mouth–shaped anastomosis. Then the vein is cut at the free end so that it is very short (1 to 2 mm), just enough for a suture line for the RIMA–vein anastomosis. The interposed vein is cut vertically and anastomosed in an oblique manner to the free RIMA, resulting in a combined LIMA–vein–RIMA (free) graft 35 to 40 cm long (Fig 1). To lessen the chance of early intimal hyperplasia and late atherosclerosis, it is important that both the LIMA and RIMA to vein anastomoses be fish-mouth–shaped and that the length of the vein in between be just enough for the oblique anastomosis.

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Table 1. Preoperative Patient Data

Patient No.	Age (y)	Angina Class[a]	Left Ventricular Function	Coronary Lesion
1	50	2	Good	Left main, three vessels
2	58	3	Good	Three vessels
3	50	3	Good	Left main, two vessels
4	57	3	Good	Three vessels
5	63	3	Good	Three vessels

[a] The classification of the Canadian Heart Association was used.

All the operations were carried out under moderate hypothermia and cold crystalloid cardioplegic arrest. First the distal end-to-side anastomoses were done with 7-0 Prolene and continuous suture technique. Then the side-to-side sequential anastomoses were completed in the same manner, ending with the left anterior descending coronary artery anastomosis (Fig 2). The diamond-shaped distal anastomoses were made as small as possible to prevent the tenting effect. As it is extremely important to prevent the rotation and kinking of the conduit, it was attached to the epicardium with several stay sutures.

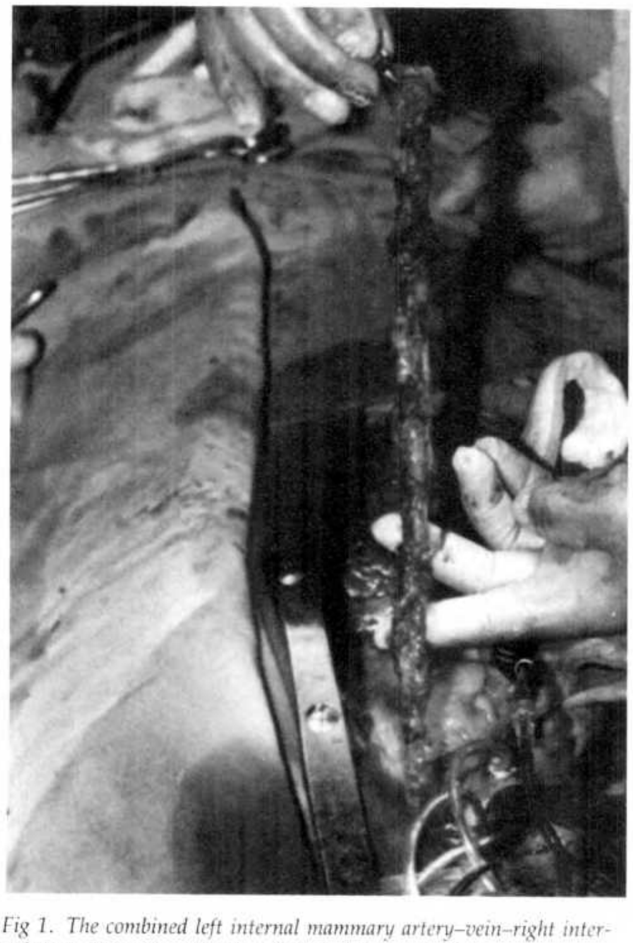

Fig 1. The combined left internal mammary artery–vein–right internal mammary artery (free) graft just after preparation.

Results

A total of 20 sequential IMA anastomoses were performed (mean number, 4 anastomoses per patient) (Table 2). There were no operative deaths. No perioperative myocardial infarctions and no sternal infections were noted. Postoperative complications occurred in 2 patients: bleeding necessitating reoperation in 1 and diaphragmatic dysfunction in the other. All the patients had an uneventful recovery without any electrocardiographic or cardiac enzyme changes.

The follow-up angiograms obtained just before discharge (tenth postoperative day) showed that all the distal anastomoses were patent (Fig 3). Exercise stress tests performed 3 and 11 months postoperatively demonstrated excellent exercise tolerance in comparison with the preoperative tests and revealed no signs of ischemia. One patient with six IMA anastomoses also had a negative thallium stress test 11 months postoperatively.

Comment

In recent years, the IMA has become the choice of many surgeons for coronary artery bypass grafting because of the enhanced long-term patency it provides. This superior late patency of the IMA compared with the saphenous vein has been demonstrated in many studies [1].

In addition, sequential bilateral and free IMA grafts have been used more frequently in an effort to maximize the number of distal IMA anastomoses. More information has become available concerning flow patterns through sequential IMA anastomoses [3, 7, 9–11]. The assumption that a single IMA can be used for more than one graft is strengthened by the findings of an increase in both IMA size and blood flow with time in several series [3, 5, 7, 9, 12, 13].

With the combined IMA graft, we also saw a substantial increase in IMA size (up to 5 mm) in all patients in the angiographic studies done 10 days postoperatively. In the patient who underwent reoperation for bleeding on the night of the operation, the graft was already seen to be more than 4 mm in diameter. This can be explained by the physiological adaptation of the IMA graft to increased myocardial demand [11, 12]. Based on the assumption that there will be an increase in myocardial demand with more distal anastomoses, the increased flow through the graft can improve the patency rate even further.

We have been using bilateral and sequential IMA grafting for coronary artery revascularization since 1985. In spite of the technical modifications developed to extend the length and thus the use of IMA grafts, it is still difficult to reach beyond the marginal branches of the circumflex and distal right coronary arteries for extensive myocardial revascularization [6, 14]. The most important question in extending the use of the IMA for the revascularization of such a large area of myocardium was whether such a conduit would be able to meet the nutritional demands of the expanded area of perfusion. The most encouraging

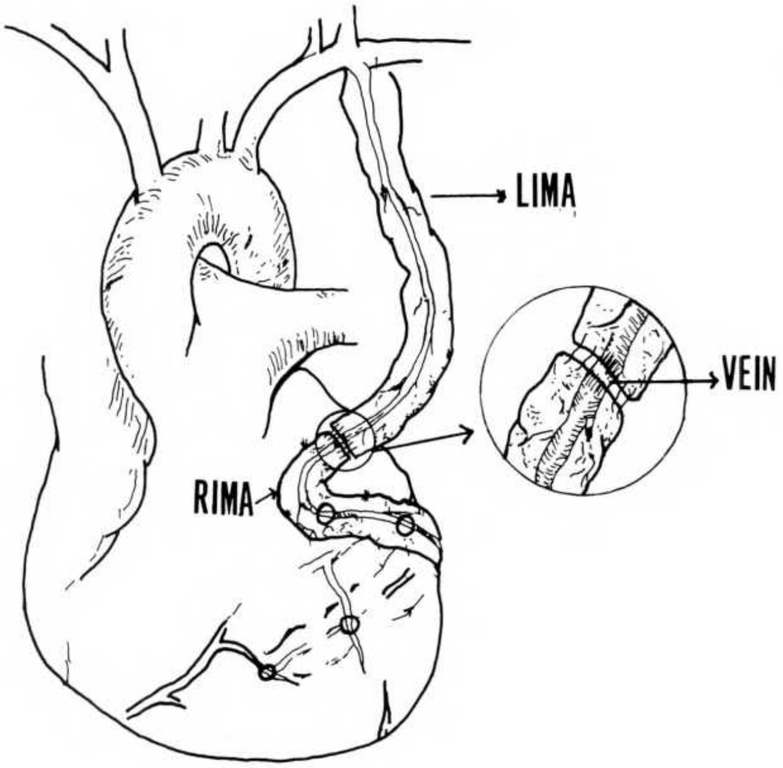

Fig 2. The completed revascularization, demonstrating the distal combined graft–coronary artery anastomoses and the left internal mammary artery (LIMA)–vein–right internal mammary artery (RIMA) (free) anastomoses.

factor that led us to this technique was our favorable results with sequential LIMA anastomoses with three distal anastomoses to the left anterior descending and circumflex coronary arterial systems. The results of the exercise stress tests and the relief of symptoms seem to support our belief that the combined IMA graft can accomplish the task of supplying this larger area.

In the construction of the new conduit, direct anastomoses between the LIMA and the RIMA were avoided because of technical difficulties. Therefore, a small piece of vein was interposed. Also, as the size of the IMA is known to increase with time and myocardial demand, the larger-caliber vein would serve to prevent narrowing in the suture line, which would occur with a direct anastomosis. The interposed vein was cut as short as possible and the LIMA–vein–RIMA anastomoses were performed in an oblique manner to minimize the risk of early intimal hyperplasia and late atherosclerosis.

Table 2. Distribution of Sequential Internal Mammary Artery Grafts

Distribution	No. of Patients
LAD–D1–D2–OM–PLRCA–RCA	1
LAD–D–OM–PLCX	2
LAD–D–PLCX–RDP	1
LAD–PLCX	1

D = diagonal; D1 = first diagonal branch; D2 = second diagonal branch; LAD = left anterior descending coronary artery; OM = obtuse marginal; PLCX = posterolateral circumflex coronary artery; PLRCA = posterolateral right coronary artery; RCA = right coronary artery; RDP = right posterior descending coronary artery.

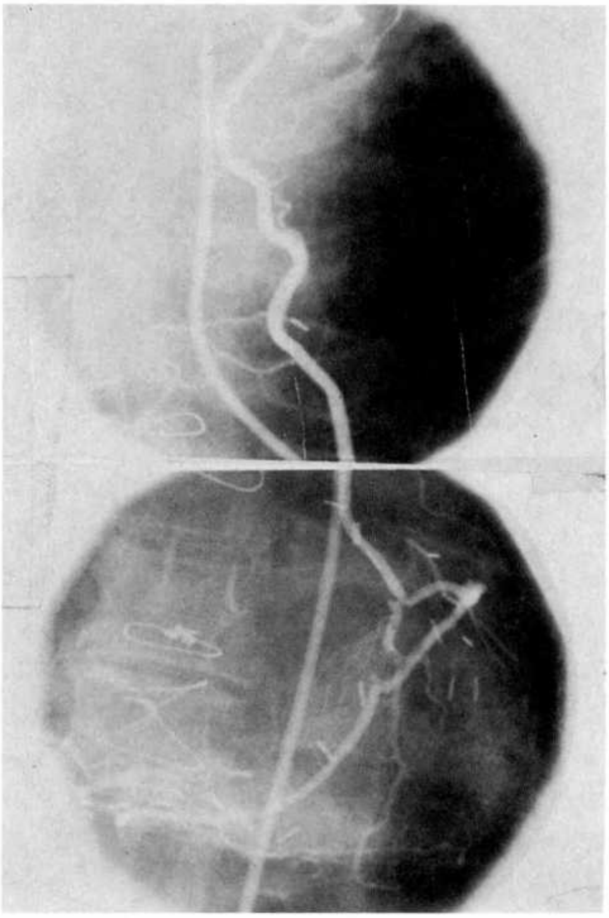

Fig 3. Angiogram made 10 days postoperatively showing patency of the distal anastomoses.

We do not recommend use of the combined IMA graft to bypass a coronary artery with a minor lesion, which could subsequently have a higher rate of competition. Nor would we use the combined IMA graft in patients with bad left ventricular function, because the prolonged period of anesthesia before the commencement of cardiopulmonary bypass can lead to ischemia, and this can adversely affect the prognosis for patients in hemodynamically unstable condition.

There are several technical considerations for use of the combined IMA graft. The vessels must be of good quality to avoid surgical difficulties. The diamond-shaped anastomoses should be as small as possible, and minor lesions should not be grafted [6, 13, 15, 16]. Diamond-shaped anastomoses are more difficult to perform, and they are made as small as possible to prevent the tenting effect.

Although this method is more time-consuming, is more technically challenging, and has certain limitations, our early results with a limited number of patients are encouraging and suggest this technique is useful in patients with multivessel coronary artery disease.

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Surgical Treatment of Cardiac Myxomas: Long-Term Results

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Between 1965 and 1988, 22 patients underwent 24 operations for cardiac myxomas. Two patients had the complex myxoma syndrome. Mitral valve replacement was required at initial operation in 2 patients. One patient died perioperatively, and 5 others died subsequently. The 16 surviving patients recently underwent evaluation at a mean duration of 9 years after operation. Ten are asymptomatic and 6 have New York Heart Association class II symptoms. Nine patients continue to be employed. Eleven are in sinus rhythm, 3 have permanent pacemakers, and 2 have chronic atrial arrhythmias. Echocardiography showed atrioventricular valve insufficiency in 3 patients and reduced contractility in 4, but no new tumor recurrences. The long-term prognosis of this relatively large group of patients with cardiac myxomas has been good. Patients without the complex myxoma syndrome had no recurrence, whereas 2 patients did require reoperation for mitral valve replacement. Long-term disability and chronic arrhythmias have been infrequent, and functional status and employability of these patients have been very good.

(Ann Thorac Surg 1990;50:557–61)

Myxomas account for approximately half of all benign cardiac tumors [1, 2]. The first successful excision of a cardiac myxoma was performed in 1954 [3]. Since then, numerous reports have described the symptomatology, diagnosis, and surgical management of cardiac myxomas [4–13]. In many of these studies, however, follow-up has been incomplete or of relatively short duration. We reviewed our experience with 22 patients who underwent operations for cardiac myxoma between 1965 and 1988 to determine the long-term results of myxoma resection.

Material and Methods

Patients

Twenty-two patients (16 women and 6 men) underwent 24 operations for cardiac myxoma at our institution from 1965 to 1988. The average patient age at operation was 50 years (range, 16 to 76 years). In 17 patients, the myxomas were solely left atrial in location: four free wall, ten fossa ovalis or septal, and three on the atrial aspect of the mitral valve. In 3 patients, the tumors were solely right atrial: one free wall, one septal, and one on the atrial aspect of the tricuspid valve. Two patients had multiple cardiac myxomas with associated bilateral adrenocortical hyperplasia. One of them had multiple right atrial myxomas with involvement of the tricuspid valve, and the other had both left and right atrial myxomas. One patient had a solitary right ventricular myxoma involving the papillary muscle of the tricuspid valve.

Fifteen patients had heart failure owing to atrioventricular (AV) valve obstruction or insufficiency and 6 had transient ischemic attacks or cerebrovascular accidents. Two patients were examined initially for repeated multiple pulmonary emboli. Two patients experienced fever, rash, weight loss, and other constitutional symptoms without definite cardiac or cerebral symptoms. Other patients, however, had fever, weight loss, and constitutional symptoms in addition to their primary cardiac complaint. The 2 patients with the complex myxoma syndrome had previously undergone bilateral adrenalectomy for hyperplasia (Table 1).

Diagnosis was made by echocardiography in 19 patients. Three patients were evaluated by cardiac catheterization only. All patients were diagnosed correctly preoperatively, although the initial diagnosis was frequently in error. Two patients were at first believed to have mitral valve disease. One additional patient was diagnosed as having constrictive pericarditis, and another was diagnosed as having rheumatic heart disease.

Physical findings were generally nonspecific, but 17 patients had an audible murmur: systolic in 13 and diastolic in 4. Only 2 patients had the classic finding of a diastolic tumor "plop."

Electrocardiographic findings included frequent nonspecific ST-T wave changes. Six of 21 patients with atrial tumors had atrial enlargement, 2 had premature ventricular contractions, and 4 had preoperative atrial arrhythmias. Eighteen patients were in normal sinus rhythm preoperatively. In 1 patient complete heart block developed, necessitating preoperative transvenous pacemaker placement. Two patients had first- or second-degree heart block before operation.

Chest roentgenogram showed cardiomegaly in 9 pa-

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Table 1. Summary of Patient Information

Patient No.	Age (y)	Sex	Location	Mode of Presentation	Length of Follow-up (y)	Present Status, Functional Class (NYHA)	Latest ECG	Latest Echocardiogram
1	52	F	RA septal and TV	CHF, weight loss	10	Died, colon CA (II)	NSR, old MI	Normal
2	16	F	RA septal	CHF, fever	24	I	NSR	Normal
3	47	M	LA septal	CHF	19	I	NSR	Normal
4	63	M	LA septal	CHF	16	II	AF, PVCs	Reduced contractility
5	56	F	LA septal	CHF, peripheral embolism, AF	11	Died, CHF (IV)	Paced rhythm, 2° AV block	Reduced contractility
6	38	F	MV	Stroke	12	I	NSR	Normal
7	62	M	LA free wall	CHF	1	Died of aortic dissection during MV replacement	1° AV block, frequent PVCs	MV regurgitation
8	16	F	LA septal × 2	Fever, rash	12	I	NSR	Normal
9	64	F	TV	CHF	12	+ Breast CA (II)	NSR	TV regurgitation
10	74	F	LA septal	CHF, AF	5	Died, CHF (IV)	AF	Reduced contractility
11	67	F	LA septal	CHF	11	Bladder CA (II)	Paced rhythm	Reduced contractility
12	63	F	LA septal	CHF	9	II	AF	Reduced contractility
13	39	F	LA septal	Stroke	9	I	Paced rhythm	TV regurgitation
14	26	F	RA free wall	Murmur (s/p bilateral adrenalectomy for hyperplasia)	7	I	NSR	Normal
	30	F	RA free wall	Pulmonary embolism, CHF	3	I	NSR	Normal
	33	F	LA, MV	CHF, fever	1	I	NSR	Normal
15	31	F	LA septal	Stroke (s/p bilateral adrenalectomy for hyperplasia)	6	I	NSR	Normal
16	31	F	MV annulus	Stroke	1 day	Died of tumor embolization during operation (IV)	NSR	. . .
17	57	M	LA free wall	CHF, ventricular arrhythmias	2.7	+ Lymphoma (I)	NSR	Normal
18	76	F	LA septal	CHF, weight loss	2.7	Died, colon CA (III)	1° AV block	Reduced contractility
19	65	F	RV, papillary muscle TV	Pulmonary embolism	2.5	I	NSR	Normal
20	57	M	LA septal	Stroke	2	I	NSR	Normal
21	75	M	LA free wall	Stroke	1.8	II	NSR	Reduced contractility
22	63	F	LA septal	CHF, heart block	1.2	II	Paced rhythm	MV regurgitation

AF = atrial fibrillation; CA = cancer; CHF = congestive heart failure; ECG = electrocardiogram; LA = left atrium; MI = myocardial infarction; MV = mitral valve; NSR = normal sinus rhythm; NYHA = New York Heart Association classification; PVCs = premature ventricular contractions; RA = right atrium; s/p = status post; TV = tricuspid valve; 1° = first degree; 2° = second degree.

tients and increased pulmonary vascularity in 12. Calcification of the tumor, although reported in the literature [12], was not noted on roentgenogram in our series.

Three patients had anemia (hemoglobin level less than 100 g/L [10 g/dL]). An elevated sedimentation rate was noted in 14 of 16 patients tested, and an elevated gamma globulin level was noted in 6 of 18 patients tested.

Cardiac catheterization was performed in 11 patients. Reduced cardiac index (less than $2.5 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$) was present in 7 of these patients. Pulmonary hypertension (mean, greater than 25 mm Hg) was noted in 6 of 11 patients. There was one complication of acute myocardial infarction secondary to tumor embolization during catheterization.

Surgical Technique

All patients underwent resection of the cardiac myxoma during cardiopulmonary bypass with core and topical hypothermia and hyperkalemic cardioplegia. Bicaval cannulation was used in most operations. Care must be taken, however, to minimize manipulation of the atrium when cannulating and passing caval tapes to avoid tumor embolization. Right atrial myxomas were resected through a right atriotomy. Left atrial myxomas were approached by the right atrium through a septal incision, except in 1 patient who underwent left atriotomy through a left thoracotomy incision. A margin of normal tissue was excised with the myxoma when possible, and septal patches were placed in 13 patients. Large free wall atrial defects were repaired with pericardial patches. Mitral valve replacement was required in 2 patients (patients 12 and 16; see Table 1) at the initial operation because the tumor stalk involved the valve annulus. One patient underwent coronary artery bypass grafting at the time of myxoma resection.

All surviving patients recently underwent physical examination, electrocardiography, and echocardiography. Most were examined at our institution, but patients living at great distances from our facilities were examined by cardiologists locally, and the studies were sent to us for review.

Results

Follow-up of patients was 100% at a mean duration of 9 years (range, 1 to 24 years) after myxoma resection.

Mortality

One perioperative death in a patient with a myxoma of the mitral valve was caused by coronary artery occlusion and low cardiac output owing to myxoma fragment embolization. Five of the 21 patients surviving the initial operation died subsequently. One patient died of congestive heart failure 5 years postoperatively at age 74 years after multiple cerebrovascular accidents. Echocardiography 3 months before her death failed to show evidence of recurrent myxoma. A second patient died of cardiac failure at age 56 years owing to myocardial infarction 11 years postoperatively. This patient was in New York Heart Association class I until approximately 6 months before her death, and echocardiography obtained before death failed to show a recurrent myxoma. A third patient died 14 months after myxoma resection while undergoing mitral valve replacement. Two patients died of noncardiac malignancies. Both had metastatic colon cancer; 1 died 32 months and the other 10 years postoperatively. In addition, 3 surviving patients have noncardiac malignancies (non-Hodgkin's lymphoma, breast cancer, and transitional cell carcinoma of the bladder).

Need for Subsequent Cardiac Operation

Tricuspid valve replacement was required for tricuspid insufficiency in 1 patient after myxoma resection (patient 19; see Table 1). The tumor had involved the tricuspid valve papillary muscle, a portion of which required resection. At the time of myxoma resection the valve was judged satisfactory. Postoperatively, however, the patient became symptomatic, necessitating replacement of the tricuspid valve. At the time of the patient's recent follow-up, she was asymptomatic and in New York Heart Association functional class I.

Mitral valve replacement for ischemic mitral regurgitation was required in 1 patient (patient 7; see Table 1) 1 year after the patient had undergone combined myxoma resection and coronary artery bypass procedure. The patient's tumor had been located in the left atrial free wall and did not involve the mitral valve. Dissection of the aorta occurred at the time of cannulation for mitral valve replacement, and the patient died. There was no evidence of tumor recurrence at autopsy.

One patient with the complex myxoma syndrome required two reoperations for myxoma recurrence. There was no tumor recurrence in patients without the complex myxoma syndrome.

Functional Assessment

All 16 surviving patients (average age, 59 years) underwent evaluation. Ten patients had New York Heart Association class I symptoms and 6 had class II symptoms. Twelve of the 21 patients surviving the initial operation were able to resume their occupations, including all of those aged less than 60 years. At the time of follow-up, 9 of the 16 patients continued to be employed. Nine patients reported use of cardiac or antihypertensive medication.

Echocardiography

Echocardiography showed mild to moderate AV valve regurgitation in 3 patients (1 with mitral valve and 2 with tricuspid valve regurgitation) and reduced contractility in 4 patients. There was no evidence of tumor recurrence.

Electrocardiography

In 2 patients with left atrial septal myxomas near the conduction system, third-degree AV block developed as a result of operation and required permanent pacemaker placement. In 11 patients new onset atrial fibrillation or flutter developed in the early postoperative period but was converted to sinus rhythm with medical therapy or cardioversion in all but 2 patients. Four patients had atrial fibrillation preoperatively. Three of these continued to have atrial fibrillation, whereas the fourth converted to sinus rhythm in the postoperative period. In 1 of the 3 patients with persistent atrial fibrillation, refractory, rapid superventricular tachycardia developed, requiring AV node ablation and permanent pacemaker placement. At the time of evaluation, 11 of the 16 surviving patients were in normal sinus rhythm, 2 had chronic atrial fibrillation, and 3 had permanent pacemakers.

Complex Myxoma Syndrome

Two patients had associated adrenocortical hyperplasia. One of these patients was aged 26 years when she first had constitutional symptoms and a heart murmur. She had previously undergone bilateral adrenalectomy for nodular adrenocortical hyperplasia and hysterectomy for a myxomatous uterine tumor. Echocardiography showed three right atrial myxomas, which were excised successfully. Four years later she had hemoptysis, heart failure, and chest pain. Echocardiography showed a recurrent right atrial free wall myxoma, which was resected successfully, with reconstruction of the right atrial free wall using an in situ pericardial patch. Two years later she again had fever, congestive heart failure, and transient ischemic attack. Echocardiography showed a new left atrial myxoma on the atrial septum, which was resected through a left thoracotomy. In addition, a small myxoma was also removed from the mitral valve annulus. The family history was significant in that a daughter and uncle had died suddenly owing to tumor embolization from atrial myxomas. Evaluation of all remaining family members failed to detect additional myxomas.

A second patient, aged 31 years and with a history of bilateral adrenalectomy for nodular adrenocortical hyperplasia, was found to have biatrial myxomas after a cerebrovascular accident. She underwent resection of both lesions and is asymptomatic at 6 years of follow-up. A recent echocardiogram showed no evidence of tumor recurrence.

Comment

Although relatively rare, myxomas are the most common primary tumor of the heart [1, 2]. Most myxomas are solitary and occur in the atria, although they may arise from the valvar apparatus (as did six of the tumors in our series).

Patients with cardiac myxomas may have varied initial symptoms depending on the location, size, and functional derangements caused by the tumor [2, 12, 14, 15]. Congestive heart failure, observed in 15 of 22 patients, is the most common initial presentation. The cause of congestive heart failure may be obstruction, valvar regurgitation, or impingement of the cardiac chamber owing to mass effect.

Neurologic symptoms, observed in 6 of our patients, are the second most frequent mode of initial presentation. These symptoms may result from thrombotic embolization or fragmentation of the myxoma. Peripheral embolization is also frequent and justifies histologic examination of all emboli in patients after peripheral arterial embolectomy.

Occasionally, constitutional symptoms are the initial mode of presentation, although this occurred in only 2 of our patients. Fever, malaise, and weight loss are common in patients with cardiac myxomas [2, 4, 14, 16, 17]. Sedimentation rate and serum gamma globulin level are frequently elevated [18]. The cause of these nonspecific findings is debatable, although the leading theory suggests an autoimmune or immunologic cause. Resolution of the constitutional symptoms and the abnormal sedimentation rate and gamma globulin level occurs after tumor resection.

Physical examination and laboratory tests in patients with cardiac myxomas generally show nonspecific findings. Often, they have an audible murmur. Classically, a tumor "plop" may be heard early in the diastolic phase and is attributed to the myxoma prolapsing into the ventricle. Only 2 of our patients had this auscultatory finding.

Echocardiography is the best method of diagnosing cardiac myxomas owing to its noninvasive nature, relatively low cost, and accuracy [2, 15, 19]. Newer diagnostic techniques such as ultrafast computed tomography and nuclear magnetic resonance are also of value in the noninvasive diagnosis of cardiac tumors [20, 21]. Studies of our most recent myxoma patient illustrate the imaging capabilities of these techniques (Fig 1). Cardiac catheter-

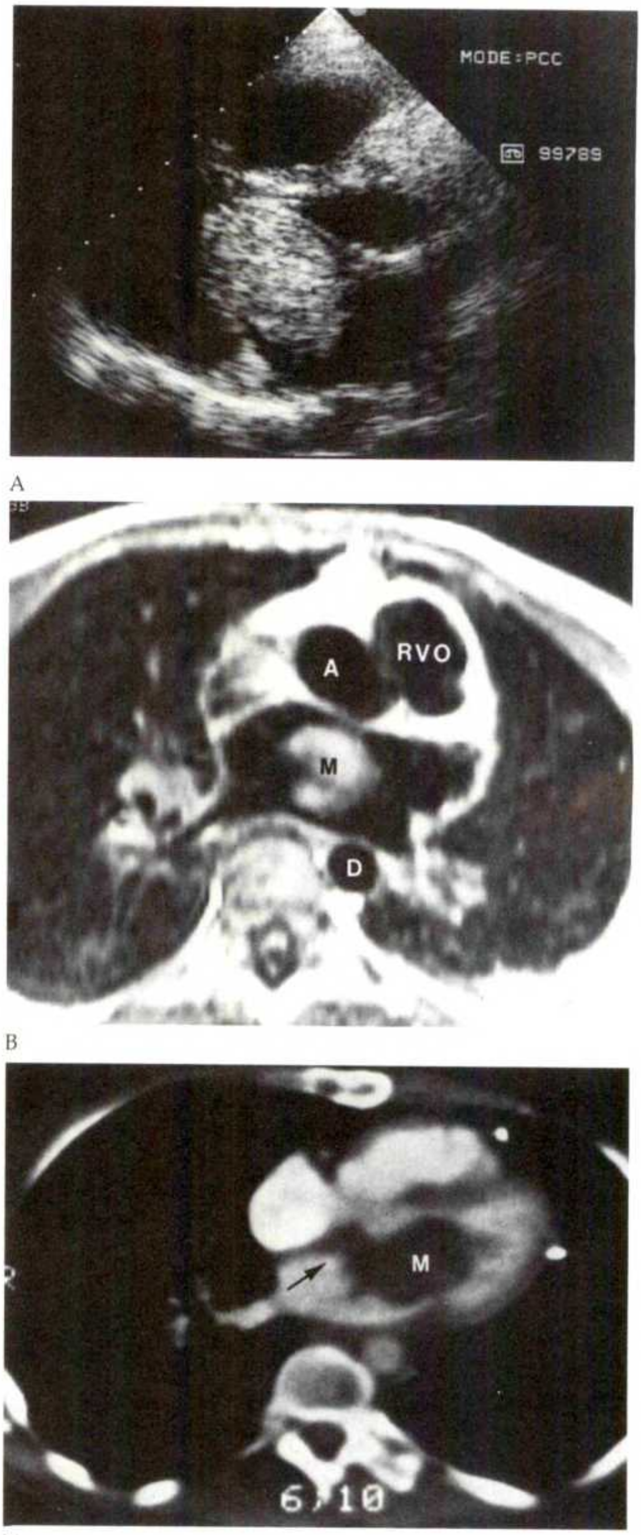

Fig 1. Imaging studies from a patient with left atrial myxoma. (A) Parasternal long-axis echocardiogram reveals the myxoma protruding into the left ventricle during diastole. (B) Gated transaxial spin-echo magnetic resonance image shows the myxoma (M) within the left atrium. (A = ascending aorta, D = descending aorta, RVO = right ventricular outflow tract.) (C) Transaxial four-chamber ultrafast computed tomographic image demonstrates the atrial myxoma (M) attached to the interatrial septum by a stalk. (Reprinted from [21] by permission of W.B. Saunders.)

ization may be indicated in select patients, but the risk of tumor embolization exists [22]. For patients with congestive heart failure or with acute neurologic symptoms and a new murmur, we suggest echocardiography as an initial diagnostic test.

Because rapid deterioration and sudden death may occur early, surgical removal is indicated once the diagnosis of cardiac myxoma is made. The approach to the heart is generally made through a median sternotomy [7], although a right or left thoracotomy may be used, especially in difficult reoperations. A variety of approaches may be taken to the left atrium. We prefer an atrial transseptal approach; alternatively, an incision posterior to the intra-atrial groove may be used. The extent of margin of normal tissue to be excised with the tumor is controversial. Although a large margin may be superior in preventing tumor recurrence on theoretical grounds [14], two studies have shown that limited resection offers low risk of recurrent disease [14, 23]. In our series, 2 patients had a microscopic positive margin. Neither patient has had recurrence after 2 and 3 years of close follow-up, respectively.

Temporary atrial arrhythmias frequently occur in the early postoperative period after myxoma resection [4]. Development of AV block most frequently occurs when the extent of resection margin is near the conduction system. Bateman and co-workers [8] studied 11 patients who underwent removal of left atrial myxomas. In all but 1 patient postoperative atrial arrhythmias developed, requiring medical treatment; third-degree AV block developed in 2 patients, requiring permanent pacemaker placement. At a mean of 48 months postoperatively, 7 of their 11 patients continued to have episodic or chronic supraventricular arrhythmias. In our series, however, there was a lower incidence of both early and late rhythm disturbances. At the time of follow-up, more than two thirds of the survivors were in normal sinus rhythm.

Two of our patients had the complex myxoma syndrome, and in 1 of these, the syndrome was familial (patient 14). Patients with familial or complex myxoma syndrome generally have symptoms at an early age and have myxomas in atypical and multiple locations [6, 24–26]. Recurrence is more frequent after surgical excision, and as complete a resection as possible should be performed to reduce this risk [6]. If a patient has either syndrome, family members should be routinely screened for myxomas by echocardiography. These patients should be closely followed for the development of recurrent tumors with annual echocardiograms.

The long-term results of this relatively large series of patients who underwent cardiac myxoma resection has been very satisfactory. There were no tumor recurrences in patients without the complex myxoma syndrome, although periodic follow-up echocardiographic examination is still recommended at this time. Functional status and employability of these patients has been very good, and long-term disability and chronic arrhythmias have been infrequent.

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Cor Triatriatum: Clinical Presentation and Surgical Results in 12 Patients

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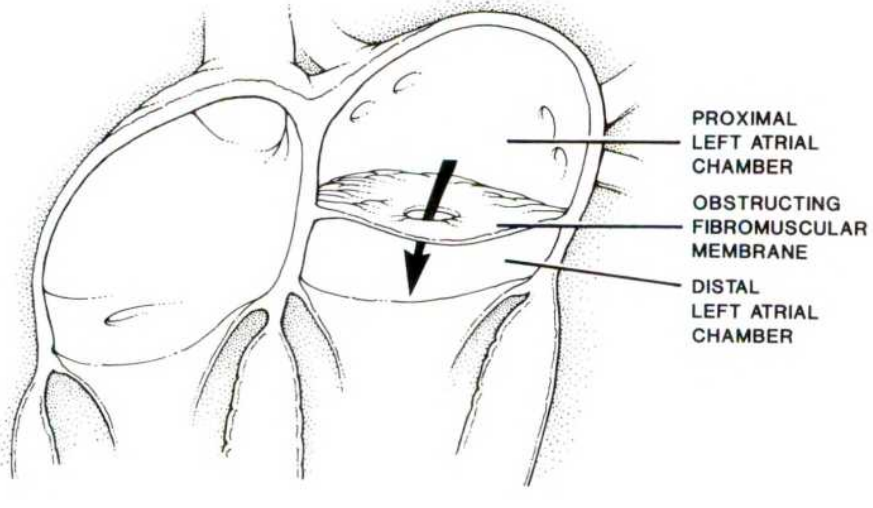
Twelve patients with cor triatriatum have been seen at our institution since 1979. The clinical presentation, diagnostic evaluation, and surgical results are outlined in this retrospective review. Operation is the treatment of choice for this rare congenital cardiac defect. One patient died 1 day before scheduled operation, and 2 patients died postoperatively, yielding a surgical mortality rate of 17% and an overall mortality rate of 25%. Resection of the obstructing atrial membrane was performed using hypothermic cardiopulmonary bypass in all cases. Left atriotomy was performed in 6 patients, and right atriotomy was performed in 7. The two postoperative deaths occurred in patients who had serious associated cardiac defects. Associated anomalies include atrial septal defect, persistent left superior vena cava, and partial anomalous pulmonary venous return. The postoperative course has been excellent in all 9 surviving patients; all remain asymptomatic. Cor triatriatum is amenable to surgical repair with excellent results when diagnosed early and when not complicated by other severe cardiac anomalies.

(Ann Thorac Surg 1990;50:562–8)

Cor triatriatum occurs in only 0.1% of children with congenital heart disease. It is characterized by the presence of a fibromuscular membrane that subdivides the left atrium. A proximal chamber most commonly receives the pulmonary veins, whereas a distal chamber typically communicates with the mitral valve and the left atrial appendage. In its most common form, this membrane is permeated by one or more small perforations, allowing blood flow directly from the proximal to distal chamber (Fig 1). Less common variations exist with the presence of an atrial septal defect in either the proximal or distal chamber. We assessed the characteristic clinical presentation, diagnosis, and surgical results in patients treated for this rare anomaly in recent years at our institution.

Fig 1. "Classic" cor triatriatum.


PROXIMAL LEFT ATRIAL CHAMBER
OBSTRUCTING FIBROMUSCULAR MEMBRANE
DISTAL LEFT ATRIAL CHAMBER

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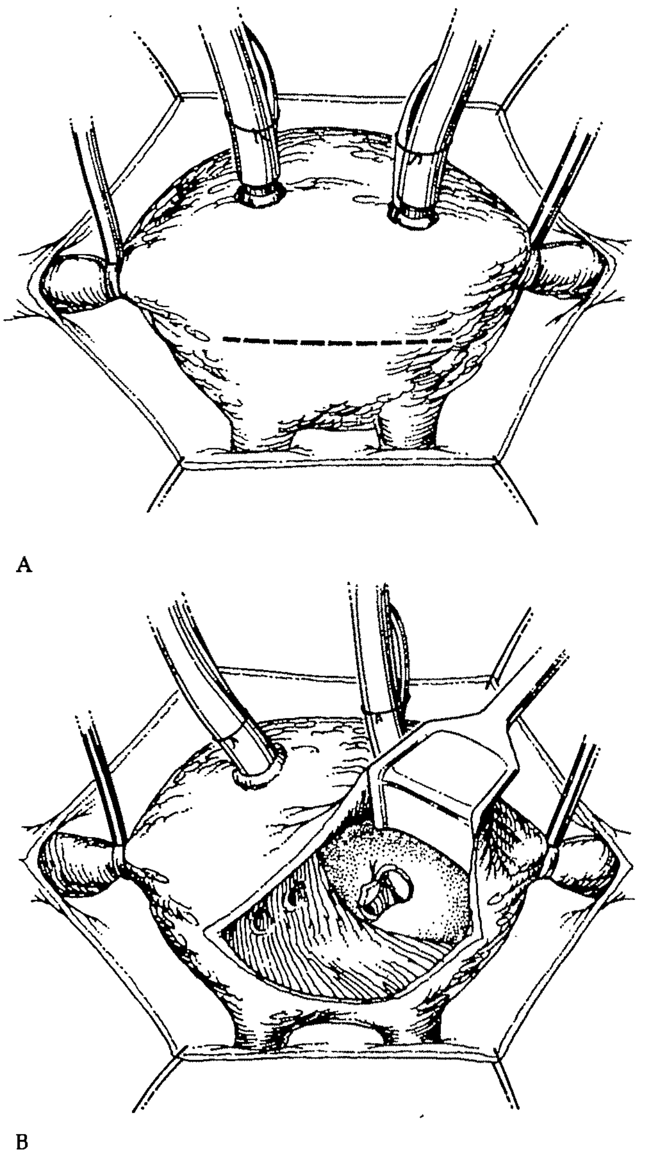

Fig 2. Surgical approach through left atriotomy. The left atrium is entered, allowing exposure of the obstructing membrane (A). After safe resection of the membrane (B), the mitral valve and left atrial appendage are visualized, and the atriotomy is closed.

Material and Methods

Patients

From 1979 to 1989, 12 patients were seen at our institution with cor triatriatum. Six were male and 6 were female (male to female ratio, 1:1). The mean age at operation was 3.1 years (range, 2 days to 15 years). Five (45%) of the patients were less than 1 year old. Preoperative symptoms ranged from mild respiratory distress to severe congestive heart failure. All patients were symptomatic preoperatively. The most frequent initial symptoms were cyanosis, fatigue, respiratory distress, and recurrent respiratory tract infection. Other commonly observed symptoms include failure to thrive and feeding difficulties. Many patients had symptoms much like those of primary pulmonary disease.

Physical examination showed a low- to medium-grade systolic murmur in all patients. Other common physical findings were moist rales, a loud pulmonary S_2 sound, and signs of right heart failure, including hepatomegaly and distended veins. Chest roentgenogram demonstrated cardiomegaly in 11 patients and increased pulmonary vascularity suggestive of pulmonary venous congestion in the same number. Two patients had questionable left atrial enlargement. Electrocardiogram demonstrated right ventricular hypertrophy and right-axis deviation in 6 patients.

All but 1 patient underwent cardiac catheterization with cineangiography. The patient who was not catheterized was a 1-month-old girl with severe congestive heart failure in whom an anomalous left atrial membrane was noted by echocardiography and subsequent emergent operation was performed. Hemodynamic data of the other 11 patients demonstrated elevated pulmonary ar-

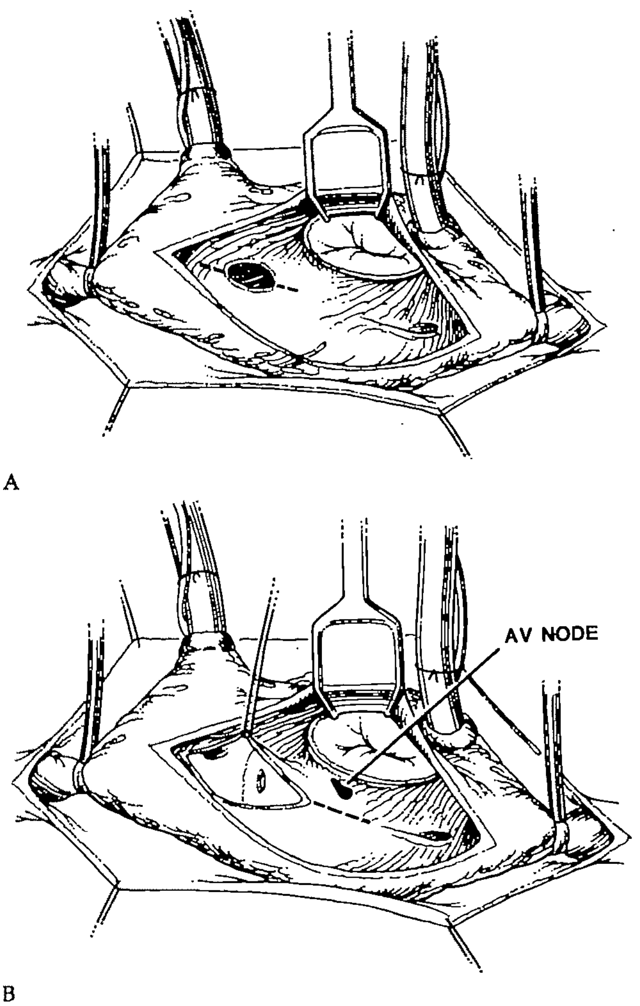

Fig 3. Surgical approach through right atriotomy. The right atrium is entered, allowing visualization of the tricuspid valve, coronary sinus, and atrial septal defect. The septal defect is then widened (A) to provide access to the left atrium and the obstructing membrane. After safe resection of the membrane (B) and visualization of the mitral valve, the septal defect is repaired, and the atriotomy is closed.

Table 1. Comparison of Surgical Results

Institution [Reference]	No. of Patients	Mean Age at Operation (y)	Patients with Severe Associated Anomalies (%)[a]	Surgical Mortality (%)	Total Mortality (%)
University of Pittsburgh, 1970 [1]	8	4.6	12.5	43[b]	50
Buffalo Children's Hospital, 1974 [2]	4	2.9	0	0[c]	0
University of Iowa/University of Pittsburgh, 1981 [3]	13	. . .	0	38	38
Children's Hospital of Michigan, 1981 [4]	6	1.8	50	17	17
Texas Heart Institute, 1983 [5]	25	6.2	48	19[d]	19
Indiana University, 1990 [present study]	12	3.1	33	17[d]	25

[a] Includes persistent left superior vena cava, single ventricle, pulmonary stenosis, transposition of the great vessels, and atrioventricular canal. [b] All deaths occurred before July 1967. [c] Reports only successfully treated cases. [d] All surgical deaths in patients with severe associated cardiac defects.

tery pressure, elevated pulmonary arterial wedge pressure, and a normal "distal" left atrial pressure. Right ventricular hypertension was noted in all patients. Oximetric evidence of a left-to-right shunt was noted in 5 patients. Cineangiography showed an abnormal left atrial membrane in 10 patients, and 2 patients demonstrated reasonable suspicion of anomalous pulmonary venous drainage. Echocardiography was performed in all pa-

Table 2. Surgical Results of 12 Patients with Cor Triatriatum

Patient No.	Age at Operation	Sex	Preoperative Diagnosis	Postoperative Diagnosis	Morphological Subtype	Procedure	Result
1	2 d	M	CT, VSD, PDA	CT, AV Canal, PDA	A	Left atriotomy, resection MB, PDA ligation, PA banding	Died of heart failure secondary to pulmonary hypertension
2	2 mo	M	TAPVR	CT, PAPVR, PLSVC	A	Right atriotomy, patch repair of right superior pulmonary vein, left atriotomy resection MB, ligation PLSVC with end-to-side anastomosis of left inferior pulmonary vein to left atrium	Died of late septic complications
3	2 mo	F	CT	Same	A	Left atriotomy, resection MB	Excellent
4	17 mo	F	CT	. . .	. . .	. . .	Died of respiratory insufficiency and cardiac failure postcatheterization
5	1.5 y	F	CT vs TAPVR, ASD	CT, ASD-OS	B	Right atriotomy, resection of tissue bridging ASD and coronary sinus ostium, patch closure of ASD with margins sutured to coronary sinus to act as a baffle	Excellent
6	1.5 y	M	CT, ASD, PLSVC	CT, PFO, PLSVC	A1	Right atriotomy, resection MB, closure PFO, ligation PLSVC	Excellent
7	15 y	M	CT	Same	A	Right atriotomy, resection MB	Excellent
8	2 mo	M	CT	Same	A	Left atriotomy, resection MB	Excellent
9	4 y	F	CT	Same	A	Left atriotomy, resection MB	Excellent
10	1 mo	F	CT	Same	A	Left atriotomy, resection MB	Excellent
11	6 y	M	CT, ASD	CT, PFO	A2	Right atriotomy, resection MB, closure PFO	Excellent
12	5.5 y	F	CT, ASD	CT, PFO	A1	Right atriotomy, closure PFO, left atriotomy, resection MB	Excellent

ASD = atrial septal defect; AV canal = atrioventricular canal; CT = cor triatriatum; MB = membrane; OS = ostium secundum; PAPVR = partial anomalous pulmonary venous return; PDA = patent ductus arteriosus; PFO = patent foramen ovale; PLSVC = persistent left superior vena cava; TAPVR = total anomalous pulmonary venous return; VSD = ventricular septal defect.

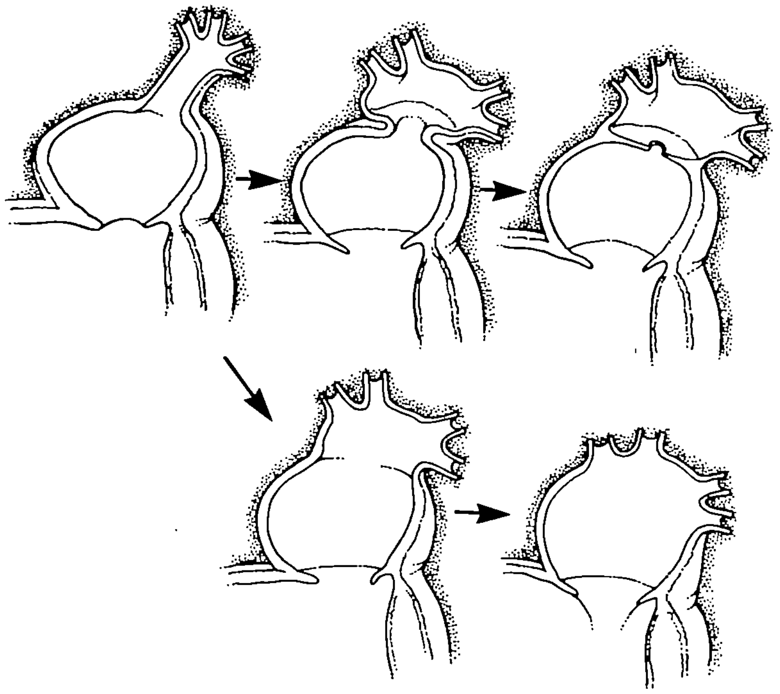

Fig 4. The malincorporation hypothesis: a failure of the common pulmonary vein to incorporate into the left atrium (top) versus normal development (bottom).

tients, and in this series has been excellent in providing a definitive diagnosis. Nine patients were noted to have an abnormal intraatrial membrane by echocardiography before catheterization or operation. One patient was diagnosed as having probable total anomalous pulmonary venous return.

Surgical Technique

All patients but 1 were treated surgically. Cardiopulmonary bypass with systemic hypothermia and cold blood potassium cardioplegia was used in all patients; all were approached through median sternotomy. The choice of atrial approach was dependent first on the suspected morphology of the defect and second on the presence or absence of associated cardiac anomalies.

Left atriotomy was preferred in uncomplicated cases. Typically, the left atrial chamber was entered superiorly and medially to the right pulmonary veins. The obstructing membrane was carefully excised, with extreme caution used to avoid damage to the mitral valve and the atrial wall (Fig 2). Six (54%) cases were approached by left atriotomy; 2 of these patients also received a right atriotomy in the same operative procedure.

Right atriotomy was preferred in cases with an associated atrial septal defect. Enlarging the defect provided excellent visualization of the obstructing membrane (Fig 3). After safe resection of the membrane, the septal defect was repaired by insertion of a Dacron patch. Seven (63%) patients were treated in this manner.

In those cases complicated by other associated cardiac anomalies, the surgical approach varied according to the nature of the defect, along with the morphology of the obstructing membrane and the presence or absence of an atrial septal defect. Six (50%) patients had associated cardiac anomalies, including atrial septal defect, persistent left superior vena cava, partial anomalous pulmonary venous return, and total anomalous pulmonary venous return. Six patients had uncomplicated cor triatriatum.

Results

Of the 11 patients who underwent surgical repair of cor triatriatum, 9 survived. The surgical mortality rate was 17%, and the overall mortality rate (including one preoperative death) was 25%. These data are comparable with those of other reported results, as shown in Table 1 [1–5]. All deaths occurred early in this series. One patient died 1 day before scheduled operation. This 17-month-old girl was admitted with symptoms of decreased exercise tolerance, diaphoresis, and fatigability. She was catheterized on the following day and was diagnosed as having cor triatriatum. Eight hours after catheterization, the patient's condition worsened, requiring ventilatory support. Two hours later, she had cardiac arrest and could not be resuscitated. Postmortem examination was not performed.

Of the two postoperative deaths, both occurred in the early postoperative period. Both patients had severe associated cardiac defects. A 2-day-old boy was noted to have respiratory distress and severe cyanosis shortly after birth. Cardiac catheterization was performed, and the infant was given a preoperative diagnosis of cor triatriatum, ventricular septal defect, and patent ductus arteriosus. At operation 1 day later, the patient was found to have a form of complete atrioventricular canal along with cor triatriatum and a patent ductus arteriosus. After right atriotomy, the anomalous atrial membrane was resected, the patent ductus ligated, and the pulmonary artery

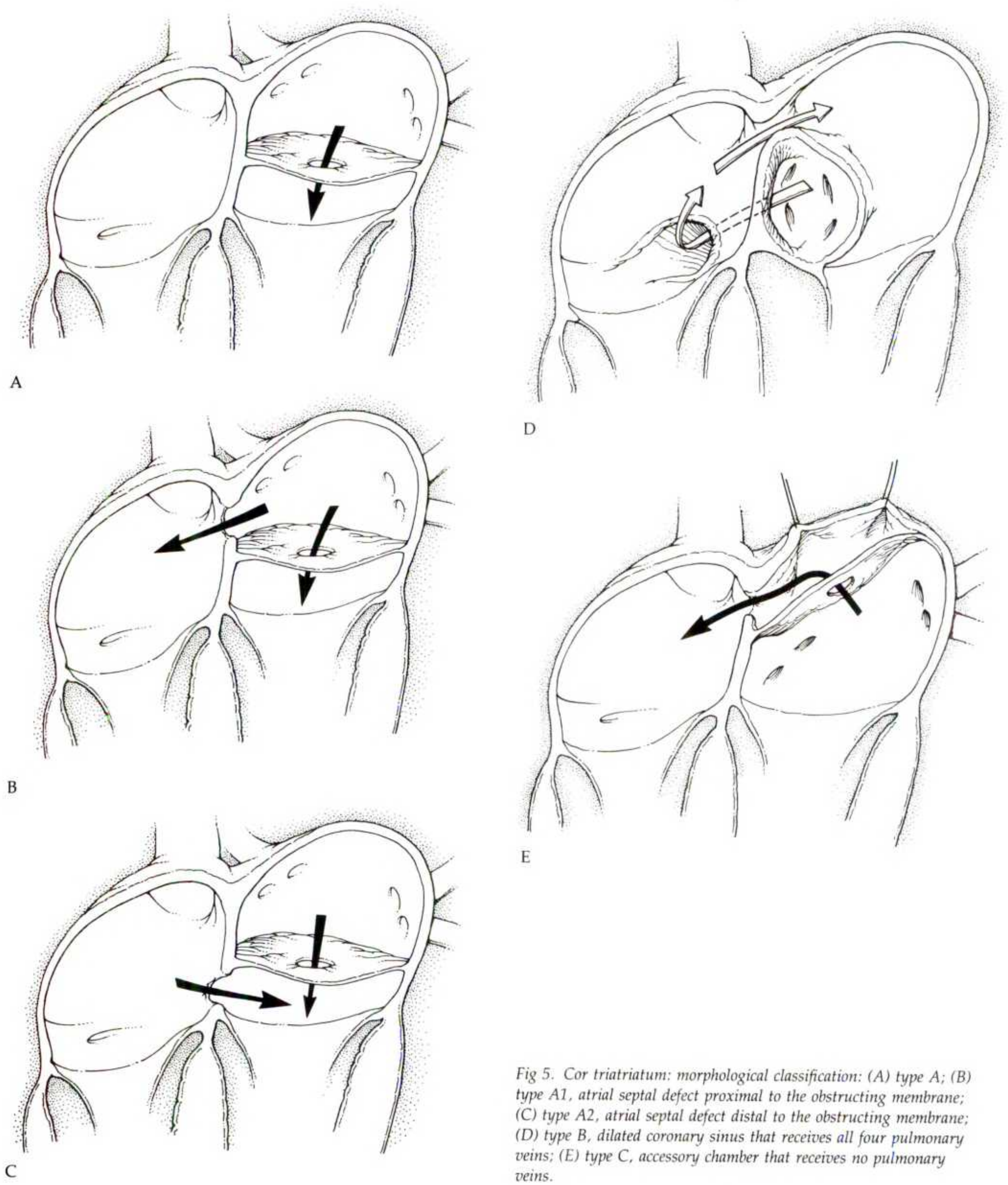

Fig 5. Cor triatriatum: morphological classification: (A) type A; (B) type A1, atrial septal defect proximal to the obstructing membrane; (C) type A2, atrial septal defect distal to the obstructing membrane; (D) type B, dilated coronary sinus that receives all four pulmonary veins; (E) type C, accessory chamber that receives no pulmonary veins.

banded. The patient was hypoxic early postoperatively, and had cardiac arrest on the first postoperative morning. The pulmonary band was removed, but the patient died several hours later.

The other postoperative death occurred in a 2-month-old boy with a preoperative diagnosis of total anomalous pulmonary venous return. At operation, cor triatriatum and a partial anomalous pulmonary venous return with a persistent left superior vena cava were found. Cardiac dilatation was marked. The postoperative course was stormy, and the patient had cardiac arrest on the night of the operation. Resuscitation, including opening of the

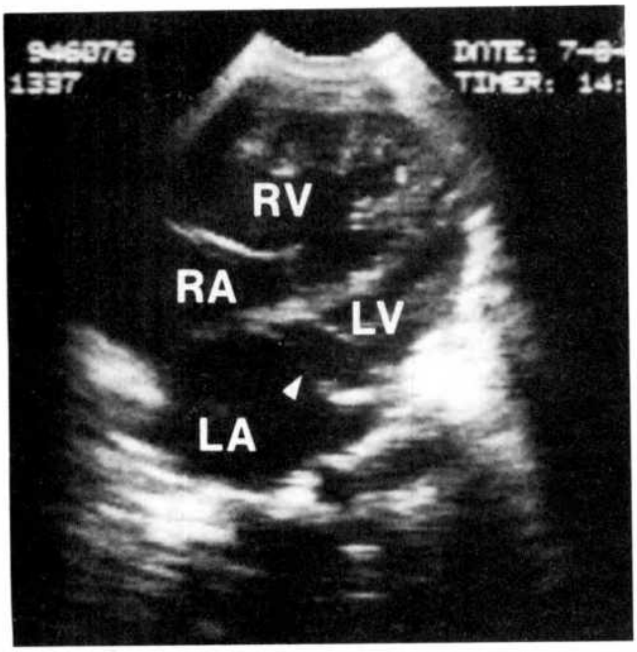

Fig 6. Echocardiogram of cor triatriatum. This four-chamber view demonstrates the presence of an obstructing membrane (arrowhead) *in the left atrium.* (LA = *left atrium;* LV = *left ventricle;* RA = *right atrium;* RV = *right ventricle.)*

mediastinal incision, was accomplished. The sternum was left open, and the skin was closed over it. The wound became infected, and surgical debridement was performed 2 weeks after the initial procedure. The patient eventually died 45 days postoperatively of septic complications.

All 9 surviving patients are in excellent health at a mean postoperative follow-up period of 1.8 years. All have had complete symptomatic relief. Electrocardiogram showed minimal right ventricular hypertrophy and right-axis deviation in 1 patient in the first postoperative year. One patient has been catheterized 11 months postoperatively, revealing overall excellent hemodynamic results. The surgical results are summarized in Table 2.

Comment

Cor triatriatum was first described by Church in 1868 [6] and subsequently has been reported only sporadically owing to its rarity. It occurs in approximately 0.1% of children who have congenital heart disease, with a male to female ratio of roughly 1:1. Successful operation for this anomaly was described in the literature at nearly the same time in 1956 by two different groups of investigators [7, 8]. The number of surgically treated patients who have since been reported is small.

Typically, a fibromuscular membrane courses diagonally in the left atrium, dividing a proximal chamber that receives the pulmonary veins from a distal chamber that communicates with the left atrial appendage and the mitral valve. Several theories have been proposed to explain the morphogenesis of this defect. The malseptation hypothesis postulates an anomalous development of the atrial septum [9]. This theory has been largely discarded because it does not account for patients with normal septal anatomy. The malincorporation hypothesis suggests an incomplete incorporation of the common pulmonary vein into the left atrium (Fig 4) [10]. Although attractive and seemingly the most widely accepted, this theory fails to explain the occasional presence of a fossa ovalis in the proximal chamber of some triatrial hearts [11]. The entrapment hypothesis integrates these two theories by stating that the anomalous membrane is the result of the entrapment of the common pulmonary vein by tissue of the sinus venous from which the septum primum develops [12]. Another group of investigators [13] has proposed impingement of a persistent left superior vena cava on the developing left atrium as a means of formation of this defect.

Many classification schemes have been proposed to describe this defect [4, 14–18]. In light of the wide range of complex morphologies, a simple, widely accepted theory is lacking. Loeffler [14] first classified cor triatriatum into three groups: no communication between proximal and distal chambers; communication through small perforations in the membrane; and incomplete membranous subdivision of the left atrium. We currently favor the following classification categories, as shown in Figure 5. Type A, or "classic" triatrial heart, consists of a proximal chamber receiving all four pulmonary veins and a distal chamber encompassing the left atrial appendage and the mitral valve; the two chambers communicate through one or more small perforations in the membrane. Depending on the presence and location of an atrial septal defect, subtypes A1 (septal defect communicating with the proximal chamber) and A2 (septal defect communicating with the distal chamber) are described. Type B hearts are those with an enlarged coronary sinus that receives all four pulmonary veins in the form of total anomalous pulmonary venous drainage [15]. Type C is an extremely rare variant in which the proximal chamber receives no pulmonary veins [17]. In our series, 7 (64%) patients had type A, 2 (18%) had type A1, 1 (9%) had type A2, and 1 (9%) had type B.

An increasing number of successfully treated cases of cor triatriatum are being reported, presumably owing to improved diagnostic procedures (ie, echocardiography) and a better awareness of the anomaly. The presenting clinical manifestations depend on the severity of the obstruction by the anomalous membrane, the morphology of the defect, and the presence of associated cardiac anomalies. Cor triatrium has been termed "supravalvar mitral stenosis," and patients have initial symptoms much like those of patients with true mitral stenosis. The diagnosis of triatrial heart must be considered in patients with symptoms of pulmonary congestion, pulmonary arterial hypertension, or pulmonary venous obstruction.

Chest radiograph typically shows signs of right ventricular enlargement, cardiomegaly, occasional left atrial enlargement, and increased pulmonary vascularity. Electrocardiographic evidence of right ventricular hypertrophy and right-axis deviation is common.

Echocardiography will demonstrate the anomaly in most patients. Cardiac catheterization is performed to confirm the diagnosis and assess associated anomalies. Most patients have elevated right ventricular, pulmonary artery, and pulmonary wedge pressures. Oximetry often signals the presence of a left to right or right to left shunt depending on the presence and location of a septal defect. Pulmonary transit time is characteristically increased. Cineangiography frequently shows an obstructive intra-atrial membrane, more so in cases in which direct communication exists between the proximal and distal chambers. In some patients, detection is difficult. Characteristically, the proximal chamber is noted to have no contractile activity, in contrast to the "true" distal chamber.

Echocardiography has been especially promising recently in the evaluation of triatrial heart (Fig 6). Our experience and the results of other investigators [19] suggests that this diagnostic tool may have greater importance and more accuracy in making a correct interpretation than do catheterization techniques. We have operated on 1 patient based solely on symptomatology and echocardiographic study. Use of magnetic resonance imaging in the diagnosis of triatrial heart has been reported recently [20]; however, in some patients, only surgical findings may provide the correct diagnosis.

Associated anomalies are a frequent complication of triatrial heart and are present in half of the patients in our series. Their presence can often preclude a correct preoperative diagnosis [21] and in our experience is associated with an increased risk of death. Operation is the treatment of choice for triatrial heart; the primary indication for operation is the diagnosis itself. The surgical approach is dictated by the anatomy of the defect and the presence of associated cardiac anomalies. Surgical results depend on the initial condition of the patient, the complexity of associated defects, and the adequacy of the repair. Cor triatriatum is a relatively simple defect to repair, and patients generally have an excellent postoperative and long-term result. Early diagnosis is of key importance and plays a major role in reducing mortality.

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Tracheal and Main Bronchial Disruptions After Blunt Chest Trauma: Presentation and Management

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Tracheobronchial disruption is one of the less common injuries associated with blunt thoracic trauma. This injury can be life threatening, however, and failure to diagnose it early can lead to disastrous acute or delayed complications. Nine cases of tracheobronchial disruption in the setting of nonpenetrating thoracic trauma were seen at four Los Angeles trauma centers between 1980 and 1987. Mechanism of injury, presentation, diagnosis, and management of these patients were reviewed. Disruptions involved the trachea in 3 patients, the right bronchus in 5 patients, and the left bronchus in 2 patients. Tracheobronchial disruptions occurred in settings of high-energy impact-type injuries and were more likely to have associated injuries than they were to occur alone. Common presenting signs included subcutaneous emphysema, dyspnea, sternal tenderness, and hemoptysis. Radiographic findings were most commonly pneumothorax, pneumomediastinum, and clavicle or rib fractures. Rigid bronchoscopy and fiberoptic bronchoscopy were both highly accurate methods for diagnosis but only in the hands of trained cardiothoracic surgeons. Delay in diagnosis increased the likelihood of postoperative complications.

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The true incidence of tracheobronchial injuries in blunt trauma is difficult to establish because many patients sustaining trauma severe enough to cause such injuries die before reaching the hospital [1]. Ecker and colleagues [2] reviewed tracheobronchial injuries over a 10-year period in Dallas County: of 27 patients with blunt tracheobronchial injuries (14 vehicular, 11 air crash, and 2 crush or fall related) only 9 (all of vehicular origin) were alive on reaching the emergency room. Bertelsen and Howitz [3], reviewing 1,178 postmortem reports of persons dying of trauma, reported that only 33 (0.03%) had tracheobronchial disruptions; 27 of them died almost immediately. Although rare, tracheobronchial injuries appear to be on the increase, associated with the increasing use of high-speed transportation [1].

For editorial comment, see page 523.

The clinical presentation of tracheobronchial injuries in blunt trauma is varied, and the initial diagnostic evaluation is often misleading. Because the emphasis in management of these injuries is on early diagnosis and intervention [1, 2, 4–7], and the acute mortality is high, timely diagnosis of these injuries is essential. We reviewed 9 patients with tracheobronchial disruption from nonpenetrating thoracic trauma. These cases were gleaned from four Los Angeles trauma centers over an 8-year period. Emphasis was placed on mechanism of injury, initial symptoms and examination, and diagnostic procedures.

Material and Methods

Records from four trauma centers in the Los Angeles area were reviewed for the period of 1980 to 1987 for patients arriving alive at the emergency room with tracheobronchial injuries from blunt trauma. The institutions included were Harbor-UCLA Medical Center, Torrance; St Mary's Medical Center, Long Beach; Memorial Medical Center, Long Beach; and Garfield Medical Center, Monterey Park, CA. Tracheal and main bronchial injuries from blunt trauma were included; no segmental bronchial injuries were found. Subsegmental bronchial injuries were excluded.

Case Reports

Patient 1

A 21-year-old female unrestrained driver arrived at the emergency room with sternal tenderness. Initial examination documented no subcutaneous emphysema, and chest roentgenogram was normal. In the ensuing hours, subcutaneous emphysema developed over the neck, with tenderness and stridor. Repeat chest roentgenogram showed pneumomediastinum. Flexible bronchoscopy showed hemorrhage into the tracheal mucosa and an abnormal tracheal ring. Results of rigid bronchoscopy performed in the operating room by the surgical resident who had performed the flexible bronchoscopy were then reported as normal, but surgical exploration performed for clinical suspicion showed complete tracheal disruption between the first and second rings. The trachea was

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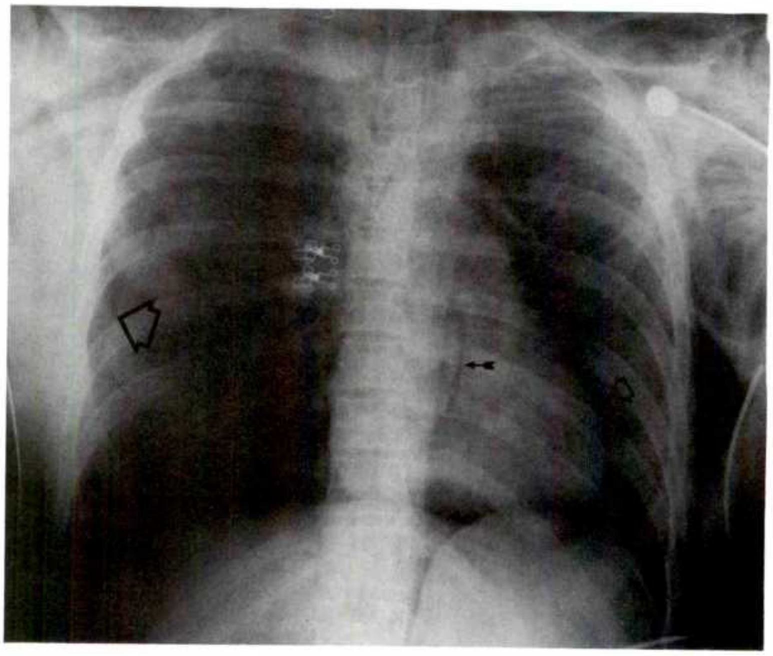

Fig 1. Chest roentgenogram of patient 3 showing a right-sided pneumothorax (large open arrowhead), *pneumopericardium* (small open arrowhead), *mediastinal air stripes* (small solid arrow), *and marked subcutaneous emphysema that outlines the pectoralis muscle fibers.*

repaired with interrupted 5-0 stainless steel sutures. The patient was discharged on the fourteenth day postoperatively with a left vocal cord paralysis.

Patient 2

A 16-year-old female unrestrained automobile passenger arrived in the emergency room obtunded, with no subcutaneous emphysema and equal breath sounds bilaterally. Chest roentgenogram showed fractures of the right second, third, and fourth ribs, a left clavicle fracture, and a widened mediastinum, but no pneumothorax or pneumomediastinum. A right thoracostomy tube was placed prophylactically, and aortography showed a right subclavian pseudoaneurysm. Subcutaneous emphysema developed shortly thereafter. Urgent repair of the pseudoaneurysm was performed through a median sternotomy. Results of postoperative flexible bronchoscopy performed by a surgical resident were reported as normal. Rigid bronchoscopy performed by the cardiothoracic surgeon, however, showed a complete right main bronchial transection, which was repaired through a right posterolateral thoracotomy using interrupted 4-0 Prolene sutures. The patient was discharged home on the sixteenth day postoperatively without complication.

Patient 3

A 20-year-old female unrestrained driver had massive subcutaneous emphysema of the face, neck, chest, and abdomen. She demonstrated hemoptysis, sternal tenderness, and diminished breath sounds on the right. Chest roentgenogram showed a right pneumothorax and massive subcutaneous and mediastinal air (Fig 1). A right thoracostomy tube was then placed, and rigid bronchoscopy showed a right main bronchial transection. This injury was repaired with interrupted 4-0 Prolene sutures through a right posterolateral thoracotomy. A transected thoracic duct was also noted. A postoperative chylothorax resolved without intervention, and the patient was discharged 3 weeks after operation.

Patient 4

A 15-year-old girl who had been ejected from her car was brought to the emergency room with complaints of chest pain and dyspnea. She demonstrated subcutaneous emphysema across the entire chest, and bilateral thoracostomy tubes were placed empirically. Chest roentgenogram showed pneumopericardium, pneumomediastinum, and bilateral pulmonary contusions. Exploratory laparotomy performed for abdominal tenderness showed a liver hematoma, right adrenal hemorrhage, and perinephric hematoma. Postoperative flexible bronchoscopy showed only main bronchial edema. A persistent thoracostomy tube air leak prompted a repeat flexible bronchoscopy 3 days after the injury, and a small left bronchial tear was noted. Bronchogram then showed a left lower lobe bronchial obstruction without extravasation. A third flexible bronchoscopy on the eighth day after injury showed a left main bronchial laceration spiraling down to obstruct the left lower lobe bronchus. This was repaired with interrupted 3-0 Vicryl sutures through a left posterolateral thoracotomy approach. Postoperative bronchoscopy 5 days after operation showed almost complete obstruction of the repaired bronchus by hypertrophic granulation tissue, which was removed initially with forceps and then again with yttrium-aluminum-garnet laser on the eleventh postoperative day. The patient was discharged home in good condition.

Patient 5

An 18-year-old unrestrained male driver reportedly crushed the steering wheel with his chest and had shortness of breath and hemoptysis. Extensive subcutaneous emphysema, Hamman's sign, and sternal hematoma were present on examination. Chest roentgenogram showed a left pneumothorax and pulmonary contusion requiring placement of a left thoracostomy tube. Flexible bronchoscopy by a cardiothoracic surgeon showed blood in the posterior carina and blood filling the left main bronchus. A bronchogram was normal. A diagnostic peritoneal lavage was positive for blood, and exploratory laparotomy disclosed splenic rupture. The patient had acute respiratory distress syndrome symptoms postoperatively, and flexible bronchoscopy by the same surgeon on the fourth postoperative day showed complete separation of the membranous trachea and main bronchi from the cartilaginous anterior structures. This was repaired with a running 3-0 Prolene suture technique through a right posterolateral thoracotomy with the patient on cardiopulmonary bypass. Bronchoscopy performed on the third postoperative day showed good repair. Sepsis and multiple-system organ failure developed, however, and the patient died 15 days postoperatively.

Patient 6

A 15-year-old boy on a bicycle was struck by a truck and arrived in the emergency room in respiratory distress. Examination showed subcutaneous emphysema over the chest and neck, Hamman's sign, and diminished breath sounds bilaterally. Chest roentgenogram showed a right first rib fracture, a left clavicle fracture, and bilateral pneumothoraces. Bilateral thoracostomy tubes were placed. The patient was extubated on the second day after injury, but the left lung remained atelectatic and an air leak persisted through the left thoracostomy tube. Flexible bronchoscopy performed on the fifth day after injury showed left main bronchial obstruction without any bronchial tears. A computed tomographic scan of the chest on the seventh day after injury suggested a bronchial transection by the great distance between the carina and atelectatic left lung. Repeat flexible bronchoscopy and rigid bronchoscopy showed a left main bronchial transection. Repair was performed through a left posterolateral thoracotomy, disclosing an abscess around the hilar stump with complete left main bronchial transection. The lingula and left upper lobe were separate from the left lower lobe and were resected; the left main bronchus was anastomosed to the left lower lobe bronchus with interrupted 5-0 Ticron sutures. The patient was discharged on the eighth day after operation.

Patient 7

A 30-year-old man was involved as the driver of an automobile struck by a train. The patient was unconscious on arrival at an outlying hospital, where assessment showed a right hemopneumothorax, basilar skull fracture, second cervical "hangman's" fracture, and a right parietal skull fracture. The patient was intubated, and a right thoracostomy tube was placed before the patient was transferred to a trauma center. At the trauma center, the patient was noted to have subcutaneous emphysema over his chest. Halo neck support and ventriculostomy were placed. Flexible bronchoscopy was performed on the third day of admission owing to marked worsening of the subcutaneous emphysema; this suggested a complete transection of the right main bronchus. A right posterolateral thoracotomy confirmed the right main bronchial transection, which was repaired with interrupted 3-0 Vicryl sutures. The postoperative course was complicated by a right-sided *Enterobacter* empyema, which was successfully treated with parenteral aminoglycosides and serial pleural taps. The patient was transferred to a rehabilitation facility 5 weeks postoperatively.

Patient 8

A 37-year-old woman thrown from her car was complaining of hip and back pain. In the emergency room, progressive dyspnea developed, associated with subcutaneous emphysema over the chest and neck, and she had respiratory arrest requiring emergent intubation. Chest roentgenogram showed a left clavicle fracture and bilateral pneumothoraces, which were treated with bilateral thoracostomy tubes. Flexible bronchoscopy showed a transected right main bronchus, which was repaired through a right posterolateral thoracotomy with 4-0 Vicryl sutures and reinforced with a pleural flap. The patient was discharged 3 weeks postoperatively.

Patient 9

A 22-year-old male unrestrained automobile passenger arrived at the emergency room with sternal tenderness, hemoptysis, extensive subcutaneous emphysema, Hamman's sign, and stridor. Chest roentgenogram showed right pneumothorax and pneumomediastinum. Flexible bronchoscopy showed a small posterior tear of the tracheal mucosa. The right lung, however, remained refractory to reexpansion after placement of two thoracostomy tubes. Rigid bronchoscopy showed a posterior tracheal tear as well as a large right main bronchial tear, which was revealed to be a complete transection of the right main bronchus at operation. The severity of the injury necessitated a right pneumonectomy, and bronchotracheoplasty was performed with 5-0 stainless steel wire reinforced with a pleural flap. The patient died 36 hours postoperatively of refractory right ventricular failure and progressive hypoxia with symptoms of acute respiratory distress syndrome.

Comment

The first successful primary repair of a bronchial rupture caused by blunt trauma was reported in 1947 by Kinsella and Johnsrud [8]. In 1959, Hood and Sloan [9] reported significantly improved results with prompt primary repair of these injuries. Since then, several series have demonstrated that early diagnosis and primary repair of these injuries leads to the fewest complications and best long-term results [1, 2, 4–7].

Traditionally, subcutaneous emphysema and dyspnea have been the most common initial signs. This was upheld in a recent series of 13 patients sustaining blunt tracheobronchial disruptions in which 85% had subcutaneous emphysema and 77% had dyspnea [5]. Another recent study reported subcutaneous emphysema in 4 of 5 patients and dyspnea in all 5 [6]. We found similar results

Table 1. Initial Signs and Symptoms in 9 Patients

	Patient No.									
Symptom	1	2	3	4	5	6	7	8	9	Total
Subcutaneous emphysema	X(D)	X(D)	X	X	X	X	X	X	X	9(2 D)
Sternal tenderness	X		X	X	X	X			X	6
Dyspnea	X			X	X	X		X	X	6
Hemoptysis			X		X	X			X	4
Hamman's sign					X	X			X	3

D = delayed.

(Table 1). All 9 patients had subcutaneous emphysema, although it was delayed up to 4 hours in 2. Dyspnea was an initial or early symptom in 6 patients. Also common were sternal tenderness in 6 of 9, hemoptysis in 4 of 9, and Hamman's sign in 3 of 9.

Initial chest roentgenograms (Table 2) most commonly showed pneumothorax (6 of 9); 3 patients had bilateral pneumothoraces. One patient had thoracostomy tubes placed for presumed pneumothoraces before chest roentgenogram, which would give a rate of 7 of 9 patients with pneumothoraces. Three of 9 patients had pneumomediastinum. Jones and co-workers [5] reported similar findings, with pneumothorax present in 5 of 13 patients and pneumomediastinum in 6 of 13. Grover and associates [6], however, reported pneumothorax in 4 of 5 patients and pneumomediastinum in 4 of 5 patients. Other radiographic findings in our series were rib fractures, clavicle fractures, pneumopericardium, and widened mediastinum. Only 1 patient had a completely normal chest roentgenogram.

The pathogenesis of tracheobronchial rupture in blunt trauma can be divided into three mechanisms. First, a decrease in the anteroposterior diameter of the thorax occurs, with widening of the transverse diameter. The lungs remain in contact with the chest wall and thus are pulled apart, creating traction at the carina. If this force exceeds the elasticity of the tracheobronchial tree, rupture occurs. Second, when the trachea and major bronchi are crushed between the sternum and vertebral column with the glottis closed, the sudden increase in intrabronchial pressure produced is greatest in the larger bronchi and rupture may occur. Third, rapid deceleration may result in shearing forces at the areas of fixation, namely the carina and cricoid cartilage [1]. These forces may act alone or in concert.

Table 2. Initial Thoracic Radiographic Findings in 9 Patients

	Patient No.									
Finding	1	2	3	4	5	6	7	8	9	Total
Pneumothorax			X	?	X	X	X	X	X	6
Pneumomediastinum			X	X					X	3
Clavicle fracture		X				X		X		3
Rib fracture(s)		X				X				2
Pulmonary contusion				X	X					2
Pneumopericardium				X						1
Widened mediastinum		X								1
Hemothorax							X			1
Normal	X									1

Question mark indicates patient in whom thoracostomy tubes were placed empirically.

Table 3. Injuries Associated With Blunt Tracheobronchial Disruptions

Patient No.	Associated Injuries
Patient 1	None
Patient 2	Rib fractures, clavicle fracture, subclavian aneurysm, and closed head trauma
Patient 3	Thoracic duct transection
Patient 4	Liver hematoma, right adrenal hemorrhage, right perinephric hematoma, and bilateral pulmonary contusions
Patient 5	Splenic rupture, myocardial contusion with right bundle-branch block
Patient 6	Rib fractures, clavicle fracture
Patient 7	Basilar skull fracture, parietal skull fracture, and second cervical vertebral fracture
Patient 8	Left clavicle fracture, left superior and inferior pubic ramus fractures
Patient 9	None

Because of these mechanisms, one would expect tracheobronchial injuries to occur in situations in which a large amount of kinetic energy is transferred to the body, eg, high-speed motor vehicle accidents. Our series illustrates that this is so; all injuries occurred as the result of automotive-associated trauma. All patients were unrestrained, and 3 were thrown from the site of initial impact. The magnitude of energy involved is evident in the following cases: 1 patient's car was struck by a train, another victim was flung from his bicycle by collision with a truck, and in two cases the patients crushed the vehicle's steering wheel with their chests.

These high-energy transfer situations would also be expected to yield a high incidence of associated injuries. Although the older literature shows associated injuries in only 50% of cases of tracheobronchial disruption from blunt trauma [1], a recent series reported 48 associated injuries in 13 patients [5]. We also found that tracheobronchial disruption in the setting of blunt trauma rarely occurs as an isolated injury (ie, in only 2 of 9 patients). Table 3 summarizes the associated injuries in our series.

Review of the older literature shows bronchial injuries equally divided between right and left [9]. We, however,

Table 4. Results of Fiberoptic Bronchoscopy Performed in 9 Patients

Patient No.	Endoscopist	Finding	Operative Finding
1	Surgical resident	Hemorrhage of tracheal mucosa; abnormal tracheal ring	Complete tracheal disruption
2	Surgical resident	Normal	Right bronchial transection
3	CT surgeon	Right main bronchial transection	Same
4			
i	Trauma surgeon	Main bronchial edema	. . .
ii[a]	Trauma surgeon	Small left bronchial tear (nonsurgical)	. . .
iii[b]	Pulmonologist	Left main bronchial laceration spiraling down to obstruct left lower lobe bronchus	Same
5			
i	CT surgeon	Blood in posterior carina, blood filling left main bronchus	. . .
ii[a]	CT surgeon	Complete separation of the membranous trachea and bronchi from the anterior cartilaginous structures	Same
6			
i	Pediatric intensivist	Left main bronchial obstruction without bronchial tear	. . .
ii[a]	CT surgeon	Left main bronchial transection	Same
7	CT surgeon	Ruptured right upper lobe bronchus takeoff and right main bronchial tear	Same
8	CT surgeon	Transected right main bronchus	Same
9			
i	ER physician	Small posterior tear of the tracheal mucosa	. . .
ii[a]	CT surgeon	Posterior tracheal tear and right main tear	Same, except that right main bronchial tear was a complete transection

[a] Second bronchoscopy. [b] Third bronchoscopy.

CT = cardiothoracic; ER = emergency room.

found right bronchial injuries in 5 of 9 patients and left bronchial injuries in only 2 of 9 patients; although this represents a fairly small sample, the right-sided preponderance agrees with the findings of another recent series in which 7 of 13 tracheobronchial injuries involved the right bronchus and none involved the left [5].

Table 4 summarizes our experience with flexible bronchoscopy. Although only 3 of 9 of the initial fiberoptic bronchoscopies were correct in their diagnosis, 5 of the 6 incorrect endoscopies were performed by other than cardiothoracic surgeons. Of 14 total flexible bronchoscopies performed on nine lesions, seven were correct in diagnosis and seven missed the lesion. Six of the seven correct endoscopies were performed by cardiothoracic surgeons, and the seventh was performed by a pulmonologist. Of the seven flexible bronchoscopies performed by cardiothoracic surgeons, six correctly identified the lesion. The one lesion missed by a cardiothoracic surgeon was later identified by the same surgeon, suggesting that it changed to a more easily identifiable form. This concept that lesions could evolve may speak to the issue of the higher accuracy of the cardiothoracic surgeons in their endoscopies, as the cardiothoracic surgeons performed the later endoscopies in some cases. The high accuracy of all bronchoscopies performed by the cardiothoracic surgeons, whether initial or delayed, however, remains impressive.

Rigid bronchoscopy was performed in 5 of the 9 cases (Table 5) and showed a lesion requiring operation in 4 of these. The one endoscopy that missed the lesion was performed by a surgical resident. Thus, rigid endoscopy in the hands of a cardiothoracic surgeon provides a 100% sensitivity, specificity, and accuracy. Bronchography did not assist the diagnosis in either of the cases in which it was used.

These results clearly indicate that the most experienced individuals are the ones most likely to make the diagnosis of tracheobronchial disruption using rigid or flexible bronchoscopy. When the patient's clinical condition suggests tracheobronchial disruption, bronchoscopy should be

Table 5. Results of Rigid Bronchoscopy Performed in 5 of 9 Patients

Patient No.	Endoscopist	Finding	Operative Finding
1	Surgical resident	Normal	Complete tracheal disruption
2	CT surgeon	Complete right main bronchial transection	Same
3	CT surgeon	Right main bronchial tear	Same
6	CT surgeon	Left main bronchial transection	Same
9	CT surgeon	Posterior tracheal tear and right main bronchial tear	Same, except that right main bronchial tear was a complete transection

CT = cardiothoracic.

performed by persons well acquainted with the appearance of these potentially life-threatening lesions. A negative bronchoscopy by an inexperienced endoscopist, ie, surgical resident, medical or pediatric intensivist, trauma surgeon, or emergency room physician does not rule out the diagnosis.

Of the 9 patients in our series, 5 were diagnosed promptly and 4 had a delayed diagnosis. Of the 5 patients diagnosed promptly, 4 had excellent surgical outcomes. The only complication was that of a unilateral vocal cord paralysis, which may have been present before operation. The fifth patient died as a direct result of the severity of his injury, which dictated pneumonectomy in a trauma setting with its attendant high mortality [10, 11]. Delay in diagnosis was detrimental in all 4 patients. In 2 of the patients, empyema or hilar abscess complicated the postoperative course. The third patient had complications with hypertrophic granulation tissue obstructing the repaired bronchus. The fourth patient had a delay in diagnosis of 4 days, which contributed substantially to his pneumonia, mediastinitis, sepsis, and subsequent multiple-system organ failure.

We conclude that in individuals treating victims of high-energy blunt thoracic trauma a high index of suspicion for tracheobronchial disruptions must be maintained to detect these rare lesions. Clinical presentation including subcutaneous emphysema, dyspnea, sternal tenderness, pneumothorax, pneumomediastinum, hemoptysis, and rib or clavicle fractures should increase suspicion. Once the diagnosis of tracheobronchial disruption is entertained, rigid or flexible bronchoscopy should be performed by an individual trained and experienced in the appearance of these injuries. Early recognition and repair of these injuries clearly decreases morbidity and mortality.

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Long-Term Amiodarone Administration Protects Against Global Myocardial Ischemia

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Reports on the effects of amiodarone on cardiac function have been variable. This study addresses the effect of long-term amiodarone administration on recovery of cardiac function after a period of global ischemia. Normotensive and spontaneously hypertensive rats were used. Normotensive rats (n = 6) received 240 mg/kg amiodarone for 4 weeks, for a total of 72 ± 3 mg. Hypertensive rats (n = 6) received 500 mg/kg amiodarone for 4 weeks, for a total of 116 ± 5 mg. Final myocardial concentrations of amiodarone and desethylamiodarone were 1.85 ± 1.75 and 0.50 ± 0.61 μg/g wet weight for the normotensive rats and 1.30 ± 0.58 and 0.31 ± 0.17 μg/g for the hypertensive rats (p = nonsignificant). Equal numbers of controls received sterile saline solution for 4 weeks. The hearts were excised and perfused in a Langendorff apparatus. The results indicate that, after 15 minutes of normothermic ischemia, hearts treated with this relatively low dose of amiodarone recovered a greater percentage of preischemic work (97% ± 13%) as compared with the controls (76% ± 17%) ($p < 0.005$).

(Ann Thorac Surg 1990;50:575–8)

Amiodarone is an effective antiarrhythmic drug that has frequently been administered to patients who subsequently are candidates for cardiac operation. Since the recognition of depressed myocardial function during cardiac operations in patients treated with amiodarone [1, 2], experimental studies of the effects of amiodarone on the myocardium have yielded conflicting results [2–8].

In view of concern that amiodarone is associated with poor recovery after global arrest during cardiac operations, we undertook this study to assess the recovery from global arrest of rat hearts at 37°C pretreated with amiodarone for 4 weeks. In keeping with previous studies with rat hearts, the experiments we report used both normotensive (Sprague-Dawley) [6] and spontaneously hypertensive [6–8] rats to determine the response of both normal and hypertrophic myocardium.

Material and Methods

Hearts from spontaneously hypertensive rats and normotensive (Sprague-Dawley) rats were used (both strains obtained from Charles River Laboratories, Wilmington, MA). All animals received humane care in compliance with the "Guide for the Care and Use of Laboratory Animals" (NIH publication No. 85-23, revised 1985). These male rats each received 20 intraperitoneal injections in a 4-week period. Normotensive rats received a total of 240 mg/kg of amiodarone in the 4-week period, and the hypertensive rats with hypertrophied hearts received 500 mg/kg. The normotensive rats weighed 186 to 217 g at the beginning of the administration period and weighed 354 to 407 g (mean, 377 ± 19 g) after 4 weeks. The hypertensive rats weighed 181 to 233 g at the beginning and 246 to 310 g at the end of the 4 weeks. The normotensive rats received 72 ± 3 mg of amiodarone in the 4-week period, and the hypertensive rats received 116 ± 5 mg. Control rats, both normotensive and hypertensive, received equal-volume injections of sterile saline solution.

At the end of the 4-week treatment period, the rats were heparinized and anesthetized with ether. The heart and great vessels were rapidly excised and placed in ice-cold heparinized saline solution. The heart was mounted on a Langendorff apparatus [9] in a warm water-jacketed (37°C) chamber (Fig 1) and perfused in a retrograde fashion at 82 cm H_2O pressure through the aorta with oxygenated (95% O_2, 5% CO_2) Krebs-Henseleit buffered solution at 37°C. The right heart emptied through the divided pulmonary artery. A latex balloon of the appropriate shape to conform to the left ventricular contour (ellipsoid, 7.0-mm major axis and 4.5-mm minor axis) was inserted into the left ventricle via the left atrial appendage through the mitral valve. The pressure in the balloon was zero when the balloon was filled to the diastolic size of the ventricle before insertion. The time from the excision of the heart to perfusion was less than 2 minutes. Bipolar atrial and ventricular electrogram leads were attached. The left ventricular balloon was connected to a pressure transducer to measure left ventricular pressure. The volume of saline solution within the balloon was adjusted so that left ventricular end-diastolic pressure was 10 mm Hg. Electrograms, left ventricular pressure, and the first time derivative of left ventricular pressure (dP/dt) were recorded on a Grass Instruments (Quincy, MA) polygraph.

After an initial perfusion period of 15 minutes, measurements of heart rate, left ventricular developed pressure, and dP/dt were made at 15-minute intervals (experimental times 0, 15, and 30 minutes). From time 30 to 45

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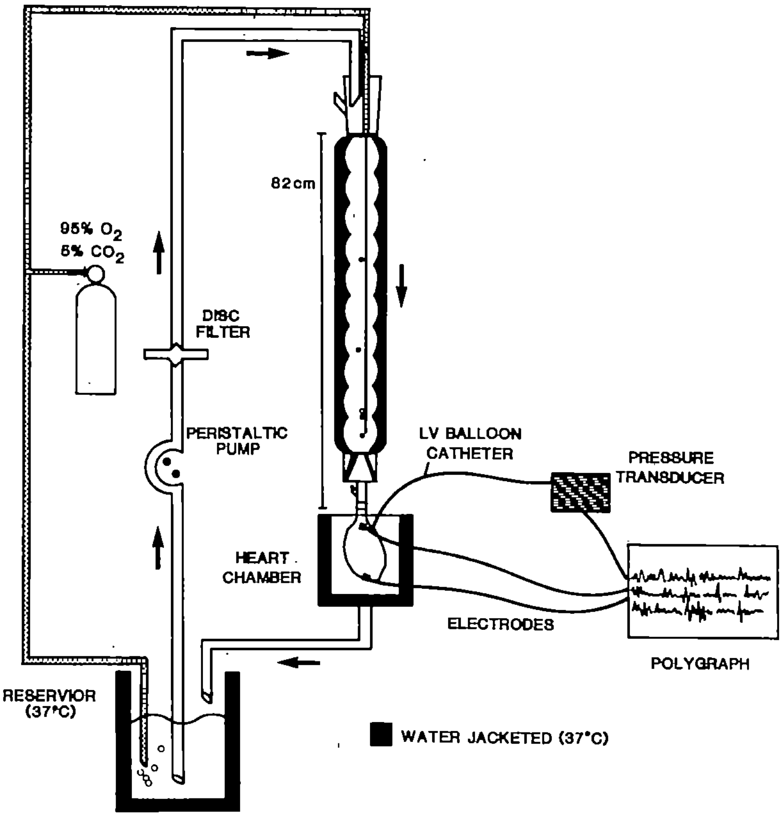

Fig 1. Langendorff apparatus: experimental setup. (LV = left ventricular.)

minutes, the aortic cannula was clamped, establishing a 15-minute period of normothermic (37°C) ischemia. At experimental time 45 minutes, the clamp was removed and perfusion was restarted. If sinus rhythm was not established spontaneously, the heart was defibrillated with a sharp tap on the apex of the ventricle. Measurements of heart rate, ventricular pressure, and dP/dt were repeated at experimental times 60, 75, and 90 minutes. Thus, the last measurements were recorded 45 minutes after start of reperfusion.

Hearts from 6 control animals and 6 amiodarone-treated animals were studied from both the normotensive and spontaneously hypertensive groups (24 animals total).

At the conclusion of each experiment, myocardium was macerated in saline solution and centrifuged. The supernatant was stored in liquid nitrogen and analyzed using high-performance liquid chromatography for amiodarone and desethylamiodarone.

Cardiac work, as expressed by the rate–pressure product, was calculated from the data. Recovery of preischemic rate–pressure product was calculated for each heart and expressed as a percentage of the preischemic value for the three measurements during the postischemic reperfusion period (experimental times 60, 75, and 90 minutes). The percentage was calculated by using as the denominator the average value of rate–pressure product at experimental times 15 and 30 minutes (preischemic). Statistical significance was determined with the t test procedure available on the Statistical Analysis Systems (SAS) software package, comparing percent work recovery of the amiodarone-treated animals with that of the controls at the same reperfusion time. The results were also analyzed with analysis of variance and Bonferroni's methods; statistically significant differences were demonstrated with all of these techniques. The p values from SAS are reported. All data are presented as mean ± standard error of the mean.

Results

At the end of the 4-week treatment period, the myocardial concentrations of amiodarone and desethylamiodarone were 1.85 ± 1.75 and 0.50 ± 0.61 μg/g wet weight for the normotensive rats and 1.30 ± 0.58 and 0.31 ± 0.17 μg/g wet weight for the hypertensive rats. This difference in myocardial concentration did not reach statistical significance.

Heart rate, left ventricular developed pressure, dP/dt, and rate–pressure product were not significantly different during the preischemia period (at 15 and 30 minutes) between the amiodarone-treated and control groups (Table 1). This was true of hearts from both normotensive Sprague-Dawley and spontaneously hypertensive rats. Both the normotensive and hypertensive hearts recovered similar percentages of rate–pressure product after the ischemic period. Hearts of amiodarone-treated rats recovered larger percentages of rate–pressure product than did those of controls, however. This was true of both normotensive and hypertensive animals. Combining the normotensive and hypertensive controls and the normotensive and hypertensive amiodarone-treated hearts showed a significant difference in recovery of function of the two groups at each of the three postischemic measurement periods ($p < 0.005$) (Table 2). Treated hearts recovered

Table 1. Comparison of Experimental Values

	Amiodarone (n = 6)					Control (n = 6)				
Time (min)	HR (beats/min)	LVDP (mm Hg)	dP/dt ([mm Hg/ s] · 10)	RPP ([mm Hg/ min] · 100)	RPP (% recovery)	HR (beats/min)	LVDP (mm Hg)	dP/dt ([mm Hg/ s] · 10)	RPP ([mm Hg/ min] · 100)	RPP (% recovery)
Sprague-Dawley rats										
15	330 ± 25[a]	81 ± 13	290 ± 74	268 ± 59		358 ± 45	97 ± 12	343 ± 73	345 ± 57	
30	338 ± 33	77 ± 11	292 ± 79	264 ± 54		350 ± 38	90 ± 8	323 ± 81	317 ± 58	
60	275 ± 43	87 ± 16	263 ± 85	241 ± 68	92 ± 24	280 ± 35	72 ± 14	208 ± 76	204 ± 62	61 ± 15
75	303 ± 37	88 ± 16	306 ± 92	267 ± 70	101 ± 17	317 ± 31	75 ± 13	238 ± 79	241 ± 58	73 ± 17
90	315 ± 38	82 ± 17	289 ± 98	259 ± 62	98 ± 15	335 ± 24	76 ± 7	254 ± 55	255 ± 27	78 ± 13
Spontaneously hypertensive rats										
15	274 ± 43	119 ± 18	535 ± 85	325 ± 65		270 ± 31	120 ± 18	583 ± 41	320 ± 30	
30	268 ± 46	114 ± 17	547 ± 53	310 ± 60		270 ± 26	113 ± 14	568 ± 68	295 ± 46	
60	247 ± 35	118 ± 30	528 ± 56	288 ± 37	95 ± 9	215 ± 62	95 ± 34	425 ± 88	195 ± 70	63 ± 24
75	259 ± 39	111 ± 16	553 ± 55	284 ± 40	93 ± 9	243 ± 42	96 ± 13	462 ± 62	231 ± 35	74 ± 13
90	259 ± 29	114 ± 13	570 ± 40	295 ± 41	97 ± 11	263 ± 32	85 ± 16	425 ± 160	225 ± 58	73 ± 21

[a] Values are shown as mean ± standard error of the mean.

dP/dt = first derivative of left ventricular developed pressure; HR = heart rate; LVDP = left ventricular developed pressure; RPP = rate–pressure product.

97% ± 13% of preischemic work by 45 minutes of postischemic reperfusion, whereas control hearts recovered only 76% ± 17% of low-pressure generation.

Comment

Both Sprague-Dawley and spontaneously hypertensive rats have been studied previously to determine the effect of amiodarone on global ischemia [6–8]. To accumulate further information on the response of hypertrophic myocardium to amiodarone, hypertensive rats were included in these experiments. Spontaneously hypertensive rats have established hypertension of 190 mm Hg when they are the size of the rats in these studies and have heart weights averaging 15% greater than normotensive rats of the same weight [10].

The total dose of amiodarone administered in these experiments was 72 ± 3 mg to rats with normal myocardium and 116 ± 5 mg to rats with hypertrophic myocardium. These dosages were derived from our preliminary work, which showed that twice the amount of amiodarone was required in hypertensive rats to achieve similar myocardial concentrations in hypertrophic and normal hearts. Mitchell and colleagues [6] administered 1,498 to 1,570 mg/kg of amiodarone intraperitoneally to spontaneously hypertensive rats for up to 20 weeks in an experiment on the effect of amiodarone on global ischemia, while the parallel group of Sprague-Dawley rats received 826 to 986 mg/kg, a difference in dosage similar to ours; they did not report myocardial amiodarone concentrations.

Table 2. Rate–Pressure Product: Percentage of Recovery in Normotensive and Hypertensive Groups Combined[a]

Experiment Time (min)	Reperfusion Time (min)	Amiodarone (n = 12) (%)	Control (n = 12) (%)	*p* Value
60	15	93 ± 17[a]	62 ± 19	<0.005
75	30	97 ± 14	74 ± 15	<0.005
90	45	97 ± 13	76 ± 17	<0.005

[a] Values are shown as mean ± standard error of the mean.

In these studies by Mitchell and colleagues [6] in normal Sprague-Dawley rat hearts, intraperitoneal administration of amiodarone for up to 20 weeks did not affect myocardial function adversely when the isolated hearts were perfused in a Langendorff preparation, but heart rate and aortic output decreased during reperfusion after hypothermic global ischemia as compared with controls. Hypertrophic hearts from spontaneously hypertensive rats pretreated with the larger doses of amiodarone had a decrease in heart rate, aortic output, coronary flow, and cardiac output, and a decrease in blood pressure as compared with control hypertrophied hearts. After hypothermic global ischemia, however, hypertrophied hearts from both amiodarone-treated and control hypertensive rats had a similar decrease in heart rate, cardiac output, and aortic output, indicating that amiodarone did not affect postischemia function adversely.

To determine a possible protective effect during global ischemia, amiodarone was administered to isolated perfused hypertrophic rat hearts at the time of initiation of either normothermic or hypothermic global ischemia; it was shown to protect the myocardium during the reperfusion period, as compared with control hearts [7, 8]. An optimal dose of amiodarone was shown to be maximally effective, whereas smaller or larger doses yielded less recovery of aortic output. In experimental coronary occlusion, infarct size was smaller in dogs receiving amiodarone intravenously after coronary artery ligation than in control animals receiving saline solution [11].

MacKinnon and co-workers [2] demonstrated a reduction of dP/dt and stroke work after 14 days of oral amiodarone administration to dogs. Landymore and associates [3], using the same oral dosage regimen, also reported lower dP/dt and stroke work, as well as failure to recover heart function after 30 minutes of normothermic global ischemia in dogs. Neither of these reports included myocardial amiodarone concentrations. Spotnitz and colleagues [4] reported mean amiodarone and desethylamiodarone concentrations in dog myocardium of 6.22 ± 0.90 and 4.14 ± 0.77 μg/mL, respectively. These myocardial concentrations of amiodarone were associated with a statistically insignificant decrease in cardiac function, although the response to inotropic drugs was less than in control dogs. Buhrman and co-workers [5] administered amiodarone orally to dogs for 4 weeks to achieve serum levels of 1 to 3 μg/mL and showed that control and treated dogs had no difference in end-diastolic pressure–volume relationships either before or after 60 minutes of hypothermic cardioplegic arrest. The response of amiodarone-treated animals to epinephrine was significantly decreased, however.

The present study suggests that amiodarone administered for a 4-week period and resulting in these tissue concentrations exerts a cardioprotective effect against normothermic global ischemia in both normotensive and hypertensive rats. Although no other studies in rats report amiodarone concentrations in the myocardium, it is apparent that long-term amiodarone administration may be detrimental to cardiac function [6]. On the other hand, amiodarone administered at initiation of global ischemia enhanced recovery during reperfusion [7, 8].

Myocardial concentrations of amiodarone in dogs [4] and humans [12] that were associated with toxicity were considerably higher than those occurring in the studies we report, suggesting that the exposure of these rats to amiodarone was less than that which occurs in most clinical cases. The low myocardial amiodarone concentrations in these animals would explain the protective effect and confirm that the negative inotropic effect of amiodarone on the myocardium is associated with larger doses. This is consistent with the suggestion that amiodarone administration be stopped in patients for as long an interval as possible before cardiac operation [13].

Why amiodarone would yield myocardial protection to a global ischemic insult is unclear. The effect of amiodarone on the myocardium is dose dependent [14, 15], and higher myocardial concentrations than in the hearts we describe would be detrimental owing to negative inotropic effects of the drug. The short duration of ischemia is less than that usually seen during a cardiac operation and may have left the myocardium responsive to the positive effects of amiodarone. Amiodarone is a coronary vasodilator [16], a slow-calcium-channel blocker [17], and a noncompetitive α- and β-adrenergic blocker [18]. Thus, circumstances of increased coronary flow and decreased myocardial oxygen consumption may have prevailed to protect the myocardium at the doses of amiodarone used in these experiments.

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Subaortic Obstruction: Intraoperative Echocardiography as an Adjunct to Operation

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Fourteen patients undergoing operation for subaortic obstruction (membranous obstruction in 11 patients, tunnel obstruction in 2 patients, obstruction due to reduplicated mitral valve tissue in 1 patient) were evaluated by intraoperative epicardial echocardiography. In all 9 patients with "discrete" obstruction who underwent prebypass epicardial echocardiography, the septal and lateral attachments of the lesion were correctly demonstrated. The precise extent of tunnel stenosis was seen in both patients. The lateral attachment of the membrane in 4 patients and multiple extensions in another 2 were identified by the epicardial study (having been missed on precordial echocardiography). The discrete membrane was enucleated in 10 of the 11 patients and was partially resected in 1. One tunnel obstruction was completely relieved; the other was partially relieved. Reduplicated mitral valve tissue in the remaining patient was completely resected. Epicardial imaging after bypass showed remnants of the membrane in 2 patients. Intraoperative Doppler echocardiography and color flow imaging confirmed the absence of clinically significant residual gradients (less than 20 mm Hg) in all but 1 patient with tunnel obstruction. Epicardial imaging provided excellent morphological information about obstructive lesions of the left ventricular outflow tract and enabled immediate assessment of surgical repair.

(Ann Thorac Surg 1990;50:579–85)

Subaortic stenosis accounts for 16% to 20% of lesions producing left ventricular outflow tract obstruction in the pediatric and adolescent age groups [1, 2]. A spectrum of pathological lesions occurs, varying from a discrete membrane to a thickened fibromuscular collar or diffuse fibromuscular tunnel [3–5], and a variety of surgical methods have been advocated for their removal [6–10]. Although often classed as congenital, some evidence shows that the membranous obstruction is acquired, probably on a substrate of congenitally abnormal ventricular myocardium [11, 12]. The natural history of unoperated lesions is that obstruction is progressive, with secondary effects on the left ventricular myocardium and aortic valve [13, 14]. There is also a tendency for the discrete membranous form to recur after operation [13]. These reported recurrences may be related in part to incomplete removal of the lesion at initial operation [5, 15].

Before the introduction of high-resolution cardiac ultrasound imaging, the diagnosis of subaortic obstruction was normally made on a combination of the clinical features and invasive cardiac catheterization findings. Left ventricular or aortic root angiography is a less than optimal technique for reliable identification of discrete fibromuscular obstruction, however. High-resolution cross-sectional ultrasound imaging has subsequently been shown to be the optimal investigative technique for determining the morphology of the left ventricular outflow tract in children [1, 16]. With the improved resolution of the current generation of cross-sectional imaging systems, it is now clear that a proportion of patients with so-called discrete obstruction can have more complex attachments of the obstructing lesion within the left ventricular outflow tract than had been realized previously. Thus, multiple septal origins and lateral insertions have been described by transesophageal studies that were not apparent from precordial ultrasound examination or at angiography [17]. A failure to appreciate the full extent of their attachments and failure to remove the entire lesion at operation may account for their recurrence.

Most patients with symptoms or signs of subaortic obstruction belong to the older age group (more than 5 years). In such patients, precordial ultrasound imaging is not always satisfactory owing to the restricted imaging window, and thus optimal morphological information may not be obtained. We therefore evaluated the use of intraoperative epicardial cross-sectional echocardiography, pulsed-wave Doppler echocardiography, and Doppler color flow mapping (1) to define the precise attachments or extension of the obstructing lesion within the outflow tract, (2) to determine the completeness of enucleation of the membrane and the relief of other types of obstruction, and (3) to determine the presence of any residual outflow tract abnormalities in the period immediately after bypass.

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Table 1. Patient Data, Operative Findings, Surgical Procedure, and Results

Patient No.	Age (y)	Findings	Operation	Associated Defects	Echocardiographic Findings After Bypass
1	4	Discrete membrane, AR	Enucleation	Partial AVSD[a]	No residual obstruction, AR unchanged
2	4	Tunnel obstruction and discrete membrane, AR	Modified aortoplasty	. . .	No residual obstruction, no AR
3	7	Discrete membrane, AR	Enucleation, PDA ligation	PDA	No residual obstruction, no AR
4	6	Reduplicated mitral leaflet	Excision, ASD closure	TGA,[a] residual ASD	No residual obstruction
5	21	Discrete membrane, AR, MR	Enucleation	. . .	No residual obstruction; AR, MR unchanged
6	21	Discrete membrane, AR, MR	Partial resection	. . .	Residual membrane, gradient < 20 mm Hg; AR, MR unchanged
7	19	Tunnel obstruction, AR, MR	Spark erosion, mitral valve repair	Severe MR	Residual obstruction, gradient 30 mm Hg, MR diminished
8	12	Septal fibrous ridge	Enucleation	. . .	No residual obstruction
9	9	Membrane, multiple septal and mitral attachments, AR	Enucleation	. . .	No residual obstruction, AR unchanged
10	3	Discrete membrane, AR	Enucleation	. . .	No residual obstruction, AR unchanged
11	2	Discrete membrane	Enucleation through VSD, VSD closure, debanding PA	VSD[a]	No residual obstruction
12	10	Septal fibrous ridge	Enucleation, VSD closure	VSD	No residual obstruction
13	8	Discrete membrane	Enucleation	. . .	No residual obstruction
14	32	Membrane, multiple septal attachments, AR, MR	Enucleation	. . .[b]	Residual membrane, gradient < 20 mm Hg; AR, MR unchanged

[a] Previous surgery. [b] Previous resection of membrane and septal myectomy.

ASD = atrial septal defect; AR = aortic regurgitation; MR = mitral regurgitation; PA = pulmonary artery; PDA = patent ductus arteriosus; TGA = transposition of the great arteries; VSD = ventricular septal defect.

Material and Methods

Fourteen consecutive patients (age range, 2 to 32 years; mean, 11 years) who were referred for operation with a diagnosis of left ventricular outflow tract obstruction due to either discrete membranous obstruction (11 patients), fibromuscular tunnel (2 patients), or reduplicated mitral valve leaflet tissue (1 patient) between January 1988 and December 1989 were evaluated by intraoperative epicardial echocardiography (Table 1). The diagnosis had been made by a combination of precordial (14 patients) or transesophageal (3 patients) echocardiography or by cardiac catheterization (4 patients). The preoperative systolic gradients (derived from continuous-wave Doppler studies) varied between 36 and 110 mm Hg.

Five of the 14 patients (36%) had associated cardiac defects requiring operation as follows: closure of a ventricular septal defect in 2 (with debanding of the pulmonary artery in 1), ligation of patent ductus in 1, mitral valve repair for severe mitral regurgitation in 1, and closure of an atrial septal defect in 1. Four patients (29%) had had previous operations: repair of partial atrioventricular septal defect in 1, Mustard procedure for transposition in 1, pulmonary artery banding in 1, and previous partial excision of a membrane and septal myectomy in 1.

Intraoperative Echocardiography

Intraoperative epicardial echocardiography was performed in a standardized manner, as described in detail previously [18]. A 5-MHz transducer was used for cross-sectional imaging and a 3.75-MHz transducer was used for pulsed- or continuous-wave Doppler and color flow studies. The transducers and wires were packed in sterile plastic sheaths; 5 to 10 mL sterile coupling gel was applied to the transducer head before packing to improve image quality. Care was taken to ensure that there were no air bubbles between the transducer head and the sterile sheath (the manner in which the transducers were packed to achieve good contact was a major factor in determining image quality). The transducers were connected to a Toshiba SSH-160A or Toshiba SSH-65A ultrasound system. A preoperative epicardial study was performed in 12 of the 14 patients (2 patients with discrete membranes did not have echocardiograms before bypass) after sternotomy and pericardiotomy but before institution of cardiopulmonary bypass. The studies consisted of scanning a fanlike series of the left ventricular outflow tract in either long-axis or short-axis view, in addition to four-chamber views. The epicardial studies were aimed at both confirming the preoperative diagnosis and obtaining precise information about the attachments of the membrane or the extent of the tunnel obstruction. Retrospective comparison of the images obtained was made with the preoperative precordial ultrasound scan.

At completion of the operation, epicardial studies were performed after bypass in all 14 patients when the hemodynamic variables had returned to near-normal, as judged by a return to near-preoperative levels of the arterial blood pressure and the atrial filling pressures. The studies were performed either by an experienced cardiologist in attendance or by the surgeon, who was familiar with the technique. All studies were continuously recorded on videotape, and both on-line and off-line analysis of the recordings was performed. In every case, the results of the off-line analysis were available to the surgeon at the time of operation. No complications resulted from the ultrasound examination.

Surgical Techniques

After median sternotomy, cardiopulmonary bypass was instituted with arterial cannulation of the ascending aorta and either bicaval (9 patients) or right atrial cannulation. Moderate hypothermia with central temperatures of 26° to 28°C was used. Diastolic cardioplegic arrest was achieved with St. Thomas Hospital cardioplegic solution administered in the aortic root after the ascending aorta was cross-clamped. Ten of 11 discrete stenoses were enucleated by blunt dissection of their attachments to the mitral valve, septum, and aortic valve; one was partially resected, leaving behind an attachment to the anterior mitral valve leaflet. No additional myotomy or myectomy was performed. One of the tunnel stenoses was corrected by enucleation of the discrete stenosis and septal resection using the aortoseptal approach [19]. The second tunnel stenosis was relieved with spark erosion of the septum. In 1 patient, reduplicated mitral valve tissue was completely excised. Thirteen of 14 corrections were performed through the aortic valve, with one of them extended to the aortoseptal approach. One discrete membrane was enucleated through a coexisting ventricular septal defect. Associated defects were repaired in 5 patients as mentioned previously.

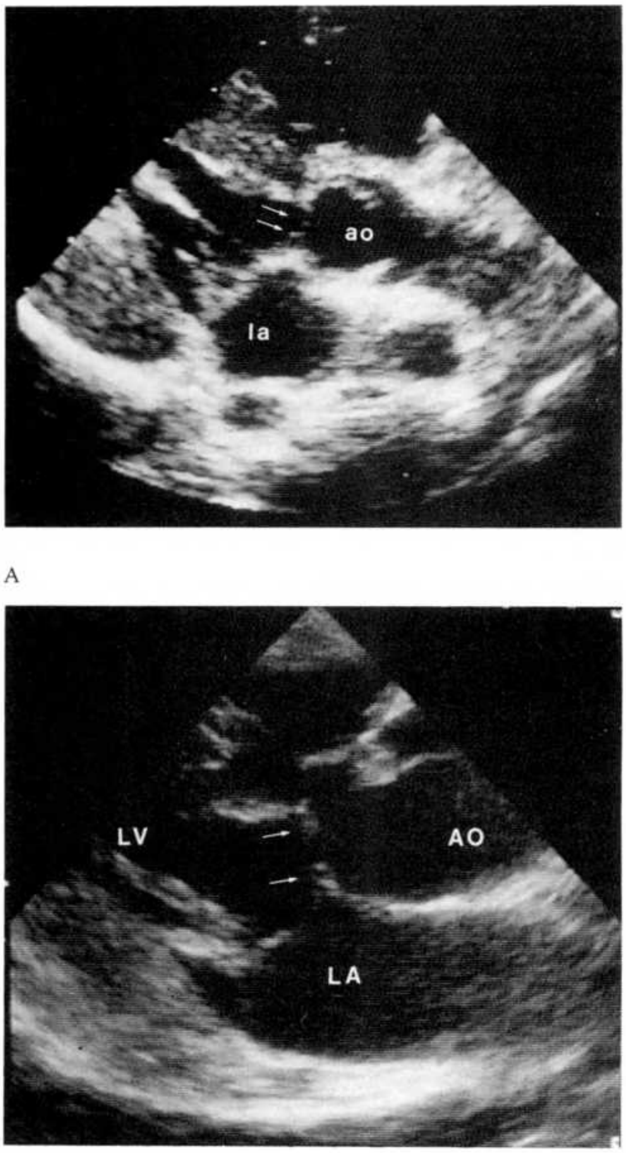

Fig 1. (A) Intraoperative epicardial cross-sectional echocardiogram showing the subaortic membrane in systole (the aortic valve is open). It is clear that the obstructing lesion is a circumferential lesion (arrows)*. The right ventricle is at the top of the picture.* (ao = *aorta;* la = *left atrium.) (B) Intraoperative echocardiogram in the long axis of the left ventricle. A discrete subaortic membrane is seen, and both septal and lateral attachments are demonstrated* (arrows)*. A downward extension of the septal attachment is also apparent.* (AO = *aorta;* LA = *left atrium;* LV = *left ventricle.)*

Results

Intraoperative Cross-Sectional Echocardiography and Color Flow Mapping Before Bypass

Excellent images of the left ventricular outflow tract were obtained in every patient. With long-axis (Fig 1) and short-axis views of the outflow tract (Fig 2) and modified four-chamber views, the precise attachments of the subaortic membrane were accurately predicted in all 9 patients with membranous subaortic stenosis who had

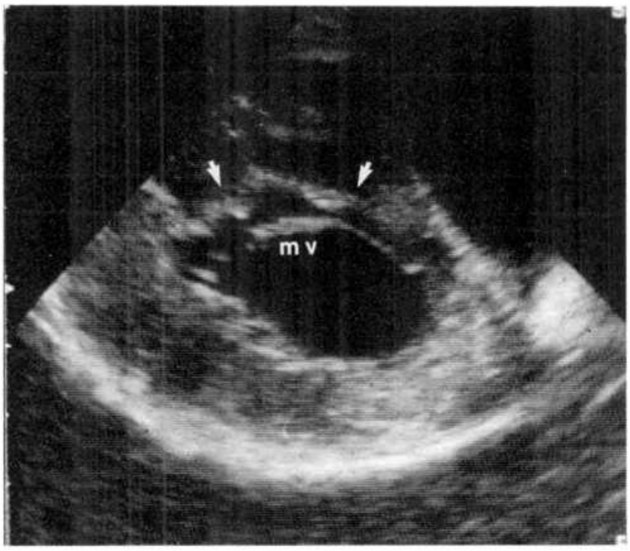

Fig 2. *Epicardial short-axis view of the left ventricular outflow tract in diastole. The entire extent of the subaortic membrane within the outflow tract, from the septum to the anterior mitral valve leaflet, is seen* (arrows). (mv = *anterior mitral valve leaflet.)*

echocardiograms before bypass. In 5 patients, the membrane was a discrete circumferential ring with septal (medial) and lateral attachments of comparable thickness; in 2 patients (one with an associated ventricular septal defect) only a septal attachment was observed. The lateral attachment varied considerably from directly beneath the noncoronary cusp of the aortic valve (1 patient) or the base of the anterior leaflet of the mitral valve in the area of mitral-aortic fibrous continuity (2 patients) to the midportion of the anterior leaflet of the mitral valve (2 patients). Two other patients had multiple extensions of the membrane: there were two separate attachments on the ventricular septum and anterior mitral leaflet in 1 patient and an extension to the aortic valve cusp in another patient.

In the patient with transposition of the great arteries and subpulmonary obstruction, precordial and prebypass imaging had shown a large mobile mass of tissue approximately 2 cm long attached along its entire length to the anterior mitral valve leaflet (Fig 3). Two separate septal attachments were observed: one directly beneath the aortic valve and a second, broader attachment lower on the ventricular septum. At operation, a reduplicated mitral valve leaflet was found, with chordal tethering to a rudimentary papillary muscle on the ventricular septum.

In 1 of the 2 patients with tunnel obstruction, the true extent of the tunnel was appreciated on epicardial imaging and the surgical procedure was modified. The aortic annulus was carefully divided between the left and right aortic cusps [19]. This enabled complete enucleation of the discrete stenosis and excision of obstructing muscle tissue. Color flow mapping of the outflow tract in patients with membranous obstruction showed systolic turbulence originating either at the site of attachment of the obstructive lesion or lower on the septal aspect of the outflow tract (2 patients), related to localized septal hypertrophy, producing additional dynamic obstruction. In both patients with tunnel obstruction and in 1 with reduplicated valve tissue, a more diffuse turbulence was observed. Mild aortic regurgitation was detected in 9 of the 14 patients, and mitral regurgitation was noted in 4 of the 14 patients (severe in 1, who required mitral valve repair).

Echocardiography After Bypass

Cross-sectional echocardiography showed that the left ventricular outflow tract was widely patent in 9 of the 11 patients after enucleation of a discrete membrane and in 1 patient after excision of reduplicated valve tissue. Pulsed- or continuous-wave Doppler studies confirmed the absence of any residual gradient in the outflow tract in every patient (including 2 patients with preoperative evidence of septal hypertrophy beneath the discrete membrane; Fig 4). Remnants of the membrane were found on cross-sectional imaging in 2 patients. In 1 patient, only the septal attachment had been excised; in another patient, with multiple attachments, a small remnant was found on the ventricular septum. In both patients, pulsed-wave Doppler studies showed peak systolic velocities of less than 2 m/s in the outflow tract (calculated gradient of less than 20 mm Hg) and these were confirmed by invasive pressure measurements. In 1 patient with tunnel obstruction who had undergone operation with the aortoseptal approach, no residual gradient was detected postoperatively. In the other patient, in whom spark erosion of the hypertrophied septal muscle was performed through the aortic valve, the calculated systolic gradient decreased from 65 to 30 mm Hg.

In 7 of the 9 patients with aortic regurgitation documented by color flow imaging in the study before bypass, the degree of regurgitation remained unchanged after bypass. In 2 patients (including 1 with tunnel obstruc-

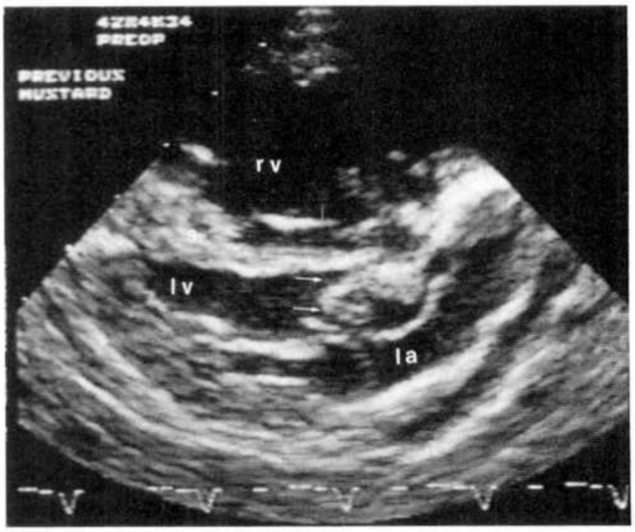

Fig 3. *Left ventricular outflow tract obstruction resulting from reduplicated mitral valve tissue. The epicardial echocardiogram demonstrated a large, mobile mass of tissue* (arrows) *with chordal attachments to the septum.* (la = *left atrium;* lv = *left ventricle;* rv = *right ventricle;* s = *interventricular septum.)*

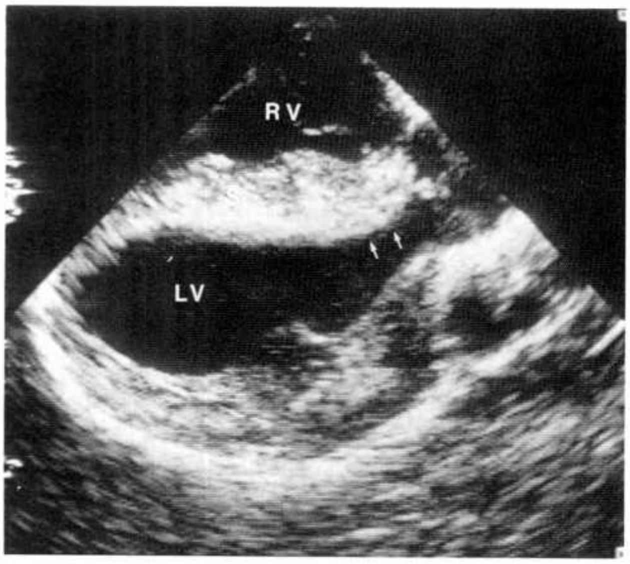

Fig 4. Postoperative epicardial cross-sectional echocardiogram of the left ventricular outflow tract after resection of "discrete" fibromuscular obstruction. There is clear evidence of subvalvar septal hypertrophy (arrows), *which was the cause of a residual mild dynamic obstruction in the patient but disappeared on follow-up.* (LV = *left ventricle;* RV = *right ventricle.)*

tion), no regurgitation was detected after bypass. Preexisting mitral regurgitation diminished in 1 patient who had mitral valve repair and remained unchanged in 3 others. Mild systolic turbulence within the outflow tract was present in all patients after bypass.

Follow-up Precordial Echocardiography

Both in patients with residual membranes on cross-sectional imaging and in 2 patients with asymmetric septal hypertrophy, repeat precordial echocardiography and Doppler studies were performed within 48 hours of operation and confirmed the findings of echocardiography after bypass. Cross-sectional echocardiography during a subsequent follow-up period of 1 to 24 months (mean, 9.2 months) has shown no recurrence of discrete membranous stenosis or change in the calculated outflow gradient in any patient.

Comment

The natural history of discrete subaortic stenosis is that in most patients it follows a progressive obstructive course over several months or years [20]. With long-standing stenosis of moderate or severe degree, there is associated left ventricular hypertrophy, which may result in secondary dynamic outflow obstruction. This in turn may account for the persistence of gradients in the outflow tract after membrane removal. The presence of a high-velocity systolic jet impinging on the aortic valve has been suggested as the cause of valve damage and regurgitation. There is also a high reported incidence of bacterial endocarditis, either on the aortic valve or on the membrane itself [13, 15, 21]. For these reasons, early surgical intervention is recommended, even in the absence of clinically significant resting gradients. Ten percent to 30% of patients have recurrence after operation, sometimes several years after the initial operation, which may be related in part to incomplete removal. Considerable debate exists over the optimal surgical technique for dealing with discrete membranous stenosis. The various proposed options include enucleation by blunt dissection [6, 7] or by sharp excision [5, 22] with or without myotomy [8, 22] or myectomy [9, 10]. More recently, balloon dilation of the discrete obstruction in both adults and children has been performed with some success [23, 24]. Although balloon dilation may be suitable as an initial procedure, only operation offers the prospect of complete removal of the lesion. In our own experience [7], it has been possible in most instances to "peel" away the membrane together with its extensions in toto; such enucleation is associated with better results [6, 7]. For the tunnel type of obstruction, myectomy is usually less effective in relieving the outflow obstruction, and clinically significant residual gradients are often present [9]. In the present series, complete relief of obstruction was obtained using the aortoseptal approach [19] in 1 patient with this lesion.

Precordial echocardiography provides satisfactory imaging of the outflow tract in most children, and usually no further investigations are required before surgical referral. In the older patient with larger chest dimensions, in whom lower frequency transducers are needed, precordial imaging does not always provide the morphological information required. Intraoperative echocardiography, however, allows the use of high-frequency transducers that may be placed directly on the epicardial surface, with concomitant improvement in image resolution. Thus, in 4 patients with discrete membranes the lateral attachment was defined only by the epicardial study; in 2 others multiple attachments that had been unsuspected during previous precordial echocardiography could be demonstrated. The ability to define the attachments or extent of obstruction is of relevance to the operative procedure as sometimes it may be difficult to evaluate the entire extent of the lesion from the transaortic approach [3, 25]. The epicardial study after bypass confirmed that the membranes had been completely enucleated in all but 2 patients, in both of whom pulsed-wave Doppler studies showed the absence of clinically significant residual gradients. Similarly, no residual gradient could be recorded in 1 patient with tunnel obstruction. There was no evidence of iatrogenic aortic valve damage, as the aortic regurgitation on color flow imaging remained unchanged in 7 patients and disappeared in 2 others as compared with prebypass studies. Where septal hypertrophy had been noted preoperatively (in 2 patients) it was clear on pulsed-wave Doppler echocardiography after bypass that the dynamic obstruction resulting from the hypertrophy was trivial, thus vindicating the decision not to perform additional myectomy.

There are potential limitations to the application of spectral Doppler techniques intraoperatively. Any assessment of residual gradients in the period immediately after bypass must be semiquantitative, as measurement of peak

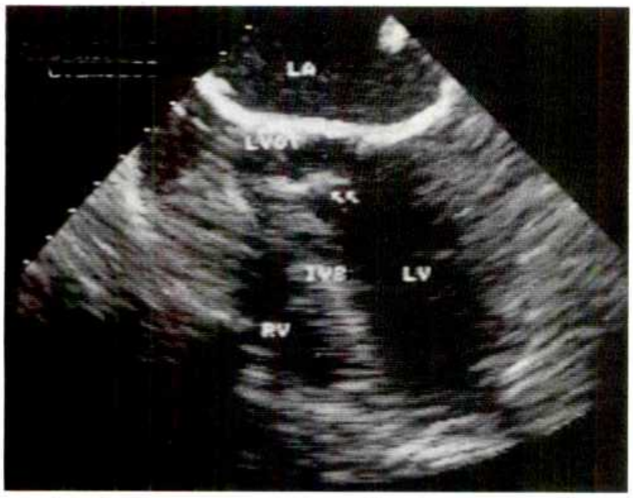

Fig 5. Transesophageal echocardiogram showing a calcified subaortic "membrane." No membrane was found at operation, and the subvalvar echoes were due to reverberations from a heavily calcified aortic valve. (IVS = *interventricular septum;* LA = *left atrium;* LV = *left ventricle;* LVOT = *left ventricular outflow tract;* RV = *right ventricle.)*

systolic velocities of flow with spectral Doppler or invasive pressure measurements are made in the face of rapidly changing filling pressures, myocardial function, and peripheral vascular resistance. Doppler color flow mapping after bypass showed systolic turbulence within the outflow tract in every patient and was not helpful in estimating the presence or degree of residual obstruction. The morphology of the outflow tract on cross-sectional imaging was a better guide in assessing the quality of the repair. Follow-up precordial Doppler echocardiography both in the immediate postoperative period and at subsequent outpatient examination have confirmed the findings of the intraoperative assessment, however.

Other potential sources of error exist with surgical referral based on noninvasive imaging techniques and are related to the limitations of image resolution even with high-frequency transducers. During the course of this study, a 30-year-old patient with symptomatic aortic valve disease (not included in this series) was referred for operation without having had previous cardiac catheterization. Precordial, transesophageal (Fig 5), and epicardial imaging had shown a heavily calcified aortic valve coexisting with what appeared to be a calcified subaortic membrane. At operation, no membrane was found, and the patient underwent aortic valve replacement alone. In retrospect, it was clear that reduplicated echoes from a calcified aortic valve had given the impression of an associated calcified subaortic membrane.

Comparison of intraoperative epicardial echocardiography and transesophageal echocardiography clearly shows that transesophageal echocardiography can be an alternative technique as an adjunct to operation for subaortic obstruction [17]. In our experience, however, higher resolution imaging can be derived from epicardial studies. In addition, Doppler evaluation of left ventricular outflow tract obstruction is not adequately reliable from within the esophagus because of possible poor alignment of the esophageal probe to the blood flow of interest. Finally, transesophageal echocardiography has only very recently become available for use in small children [26] and would not have been possible in at least 8 of the patients in the present investigation. Transesophageal echocardiography requires separate probes of smaller size, whereas the same epicardial probes can be used in patients of any age.

Intraoperative echocardiography provided excellent morphological information about the left ventricular outflow tract in patients with a variety of obstructive lesions. Complete excision of the obstruction could be confirmed "on the table." This was best judged on cross-sectional imaging. The presence of residual fixed obstruction and of aortic valve regurgitation could also be correctly predicted by epicardial pulsed- or continuous-wave Doppler examination allied with color flow mapping. Long-term follow-up studies are required to judge whether recurrences of subaortic membranes or fibromuscular obstruction are related to incomplete excision of the initial lesion, but intraoperative imaging after bypass provides a baseline that may be used for subsequent clinical or echocardiographic evaluation.

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Diagnostic Imaging and Surgical Treatment of Dumbbell Tumors of the Mediastinum

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We describe the diagnostic procedures and surgical approaches employed in 5 patients with dumbbell tumors of the mediastinum. Magnetic resonance imaging accurately described the existence and longitudinal extension of the intraspinal component of the tumor and assisted in choosing the appropriate surgical approach. Both the intrathoracic and intraspinal components of the tumor were resected at one time by a thoracic and neurosurgical team. We employed the Grillo technique three times and a separate laminectomy and thoracotomy approach. Magnetic resonance imaging proved the most useful diagnostic technique for suspected dumbbell mediastinal tumors. In our experience, the extended thoracotomy proposed by Grillo and co-workers worked well for small tumors involving only one foramen in which the intraspinal extension was limited to 2 to 3 cm, and when no more than two laminectomies were required. On the other hand, thoracotomy and a longitudinal paravertebral incision are preferable for larger tumors (more than 4 cm) involving more than one foramen in which the intraspinal extension exceeds 2 to 3 cm, for tumors requiring multiple laminectomy, and when bony infiltration is present.

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Whether neurogenic tumors of the posterior mediastinum have an intraspinal component must be suspected preoperatively [1, 2]. Although myelography [1] and computed tomography (CT) [3, 4] have been used as specific diagnostic techniques to rule out neural canal involvement, each technique has disadvantages. Myelography causes discomfort and is potentially dangerous to the patient; CT imaging is limited to the axial plane. Current practice [1, 4, 5] is to treat intraspinal lesions in combination with the intrathoracic component to avoid complications. Surgical resection of the dumbbell tumor may be performed either by two separate incisions, one for laminectomy and one for thoracotomy (according to Akwari and associates [1]), or by the combined approach proposed by Grillo and co-workers [3].

The purpose of this report is to point out the advantages of magnetic resonance imaging (MRI) in the preoperative study of neurogenic tumors of the posterior mediastinum and to evaluate the indications for each of the two proposed surgical approaches according to the size, location, and intraspinal extension of the tumor.

Methods and Results

Between March 1987 and December 1989 we operated on 14 neurogenic tumors of the posterior mediastinum, 5 of which (35%) were seen as an intraspinal extension (Table 1). The patients included 8 males and 6 females, between 16 and 55 years of age (mean, 33 years). In 7 patients, including 2 with dumbbell tumors, the presenting symptom was chest pain; no patient had motor disorders or other neurological disturbances. The diagnostic work-up included routine laboratory tests, electrocardiography, spirometry, and a neurological examination, all of which were normal for all patients. All patients underwent posteroanterior and lateral chest roentgenograms and CT scans, and MRI was performed in 6 patients; only 2 patients underwent myelography. Magnetic resonance imaging was performed after CT in the 5 patients with dumbbell tumors and in 1 additional patient whose CT suggested an intraspinal extension. The results of MRI were clear and self-explanatory (Figs 1, 2A).

Those patients with no intraspinal involvement (Fig 1) were approached by standard posterolateral thoracotomy; the intercostal space selected for access varied according to the location of the tumor (fourth to sixth intercostal space). In all cases the resection was technically easy and radical.

Three patients with small dumbbell tumors (less than 3 cm) and limited intraspinal involvement (Fig 2) were approached by the technique proposed by Grillo and co-workers [3]. An L-shaped incision was performed: its vertical paravertebral branch extended approximately 5 cm above and below the foramen to be explored, and its transverse branch swept anteriorly parallel to the sixth rib. After a skin and subcutaneous flap was created, the latissimus dorsi and trapezius muscles were divided and the chest was entered through an intercostal space selected according to the location of the tumor. After the intrathoracic lesion was resected up to its foraminal isthmus, the neurosurgical procedure was initiated. The intraspinal component was removed by a double approach: first, the foramen was carefully enlarged by a

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Table 1. Patient Data

					Thoracotomy		Laminectomy		Complications		
Patient No	Age (y)	Sex	Pain	Operation	Space	Tumor Dimension (cm)	Level	Tumor Dimension (cm)	Intraop	Postop	Histology
1	43	M	−	Grillo	Sixth	3	D-7	2	. . .	. . .	Schwannoma
2	19	M	+	Akwari	Sixth	8	D5-6-7	10	Spinal fluid leakage[b]	Meningitis	Malignant schwannoma
3	55	M	+	Akwari	Sixth	5[a]	D5-6-7	9	. . .	. . .	Myxoid chondrosarcoma
4	50	F	−	Grillo	Fifth	3	D4-5	2	. . .	. . .	Schwannoma
5	31	M	−	Grillo	Fifth	2.5	D-5	1	Spinal fluid leakage[b]	. . .	Schwannoma

[a] This patient had resection of posterior arch of sixth and seventh rib. [b] The fistula was closed by fibrin glue and an intercostal muscle flap.

bone-cutting forceps and the anterolateral component of the tumor was controlled without retracting the medulla (transthoracic, transforaminal route). After the erector spinae muscle had been dissected and retracted through the vertical branch of the skin incision, a hemilaminectomy was performed in the usual fashion. This route provided an excellent exposure for the radical resection of both the intrathoracic and intraspinal components. There were no intraoperative or postoperative problems.

For 2 patients with larger tumors (more than 3 to 4 cm) that involved more than one foramen or had infiltrated the ribs and vertebrae (Figs 3, 4), two separate incisions were made [1, 5]. The patient was placed on the operating table in a semiprone position, and the hemithorax and back from the mid-axillary line to the contralateral paravertebral line were disinfected and draped. First, a paravertebral incision was carried out by the neurosurgical team; an extended laminectomy (D5-6-7) was performed under good exposure. The whole extrathoracic component of the tumor was resected up to its foraminal isthmus. The incision was left open and posterolateral thoracotomy on the seventh intercostal space was performed with the neurosurgeon standing by. In patient 2, who had no bone infiltration, the parietal pleura and the vascular connections between the tumor and the parietes were divided, and the intrathoracic lesion was easily removed by application of gentle traction on the cut ends of the two foraminal isthmi. In patient 3 the main bulk of the tumor was similarly resected; subsequently, the posterior arch of the 7th and 8th rib was resected, and D-7 was cleaned from the tumor by an osteotome. Malignancy was confirmed histologically in both cases (malignant schwannoma and mixoid chondrosarcoma [6]).

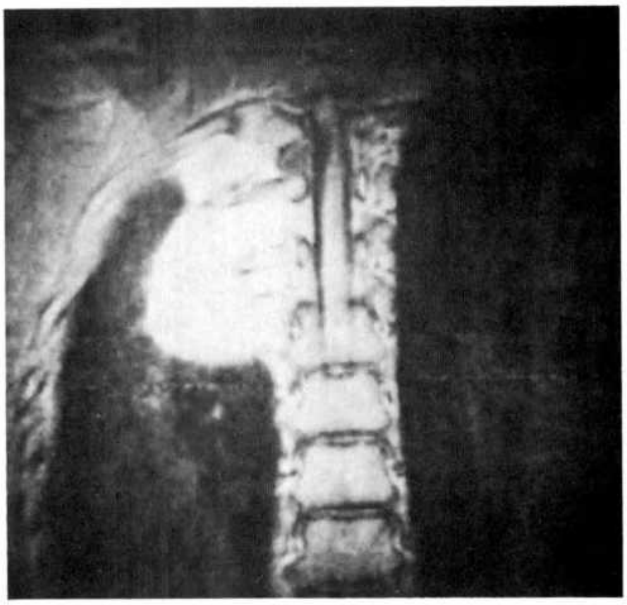

Fig 1. Magnetic resonance imaging of schwannoma of the posterior mediastinum without intraspinal involvement. The neural canal is tumor free and the medulla is clearly visible (compare with Figs 2A, 3A, 4).

In all 5 patients, the tumors were entirely extradural. There was one major complication: in patient 2 the dural space was entered and a leakage of spinal fluid occurred. The fistula was closed by fibrin glue and a muscular flap, but meningitis developed. The patient recovered fully in 30 days. All patients tolerated the procedure well and are now back to their normal activities.

Comment

Magnetic resonance imaging has been employed in recent years as an alternative to or in combination with CT for the evaluation of mediastinal tumors, but neither technique seems to offer substantial advantages [7–10]. Computed tomography provides a clearer delineation of a mass from surrounding structures [7, 8], but MRI can supply images on the coronal and sagittal planes. This latter feature makes MRI particularly valuable in evaluating neurogenic tumors of the posterior mediastinum, which may extend into the neural canal. In the past, myelography and CT have been employed in the evaluation of dumbbell tumors [1, 3]. Myelography accurately describes the length of tumor extension in the neural canal, and CT provides excellent imaging of the widening

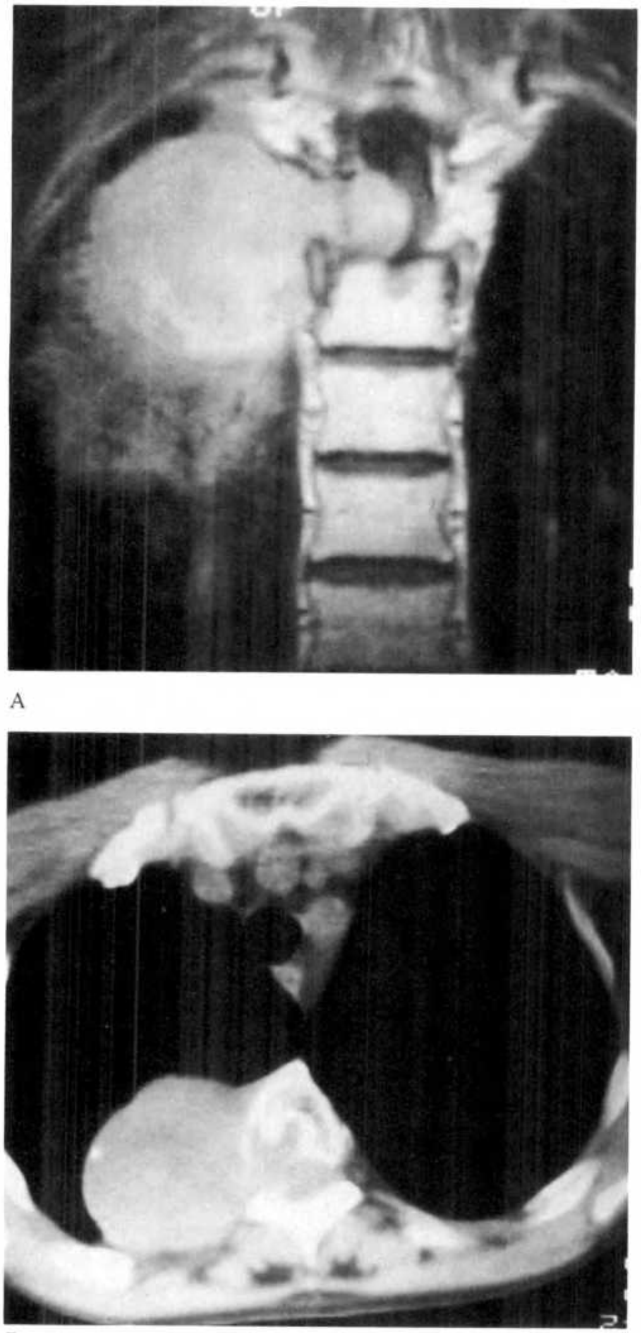

Fig 2. (A) Magnetic resonance imaging of mediastinal schwannoma with a dumbbell shape (patient 4). The extension of the tumor into the neural canal through one foramen is evident (compare with Fig 1). (B) Computed tomography of the same patient. The tumor was resected by the Grillo technique.

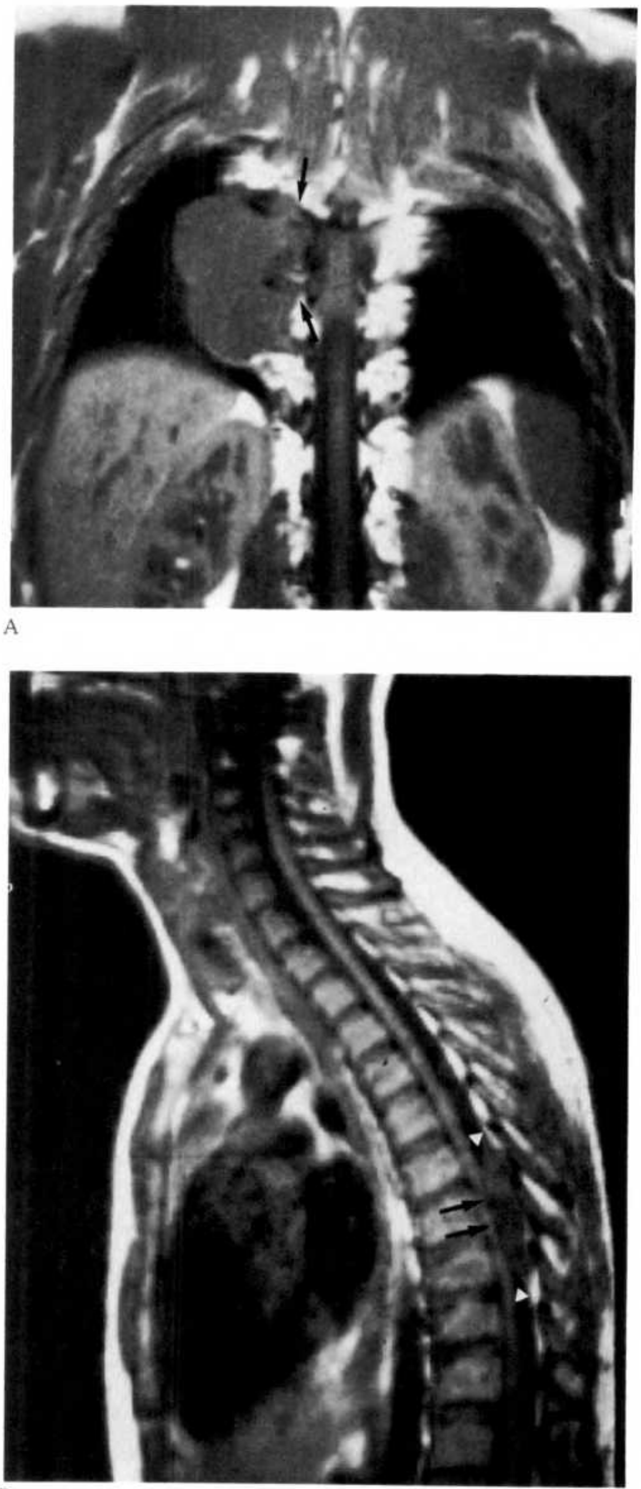

Fig 3. Magnetic resonance imaging in the coronal (A) (compare with Fig 1) and sagittal (B) planes of malignant schwannoma (patient 2). The involvement of two foramina is demonstrated by the coronal cut (A: the arrows indicate the upper and lower margins of tumor infiltration); the extension of the intraspinal involvement is precisely demonstrated by the sagittal cut (B: the white arrows indicate the upper and lower limits of the tumor in the neural canal, the black arrows indicate the area of displacement and compression of the medulla). Radical resection was carried out by the Akwari approach.

of vertebral foramina due to tumor proliferation. In our experience, CT provided the best images for the delineation of tumor margins, but MRI proved especially valuable in investigating suspected dumbbell tumors. In fact, MRI axial cuts have little less definition than CT, but the images on the coronal and sagittal planes (Figs 1, 2A, 3, 4) allow a precise evaluation of intraspinal extension of the

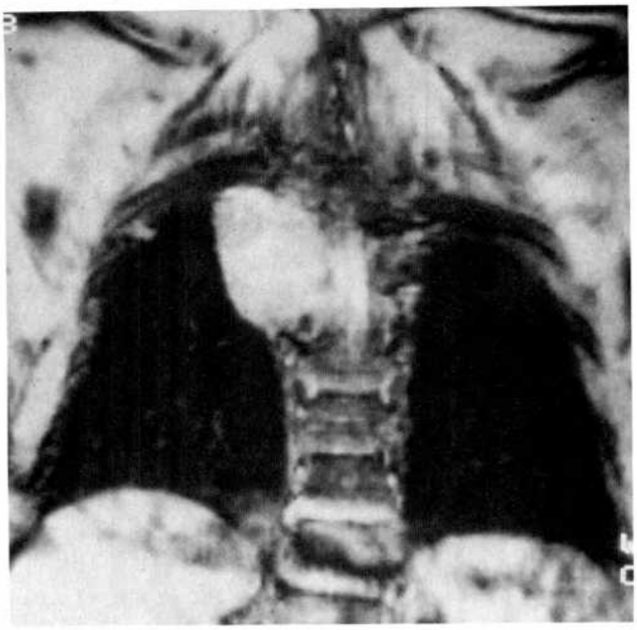

Fig 4. *Magnetic resonance imaging of myxoid chondrosarcoma (patient 3). Intraspinal and bone involvement is evident in the coronal plane (compare with Fig 1).*

tumor, thus suggesting the most appropriate surgical approach. Some authors [1] have advocated plane radiography as a means to identify the widening of the intervertebral foramen. We believe that plane radiography alone is not sufficient for the preoperative staging of a suspected dumbbell tumor; plain chest roentgenography should be complemented routinely by MRI in suspected cases.

At the present time, the surgical resection of the intrathoracic and intraspinal tumor components at one time is the procedure of choice for dumbbell tumors. Akwari and associates [1] recommend that the neural canal be approached at first through a paravertebral incision. Then a thoracotomy should be performed in the same operative session, thus avoiding serious complications such as spinal cord injury, bleeding, and medullary compression due to hematoma. In 1983, Grillo and co-workers [3] proposed a combined approach to be carried out through a posterolateral thoracotomy extended posteriorly by an upward paravertebral branch that allows access to the selected intercostal space and the vertebral laminae. Our experience suggests specific applications for each technique. When the tumor involves only one foramen and the intraspinal extension is limited, thus requiring the resection of only one or two contiguous laminae, the Grillo approach is easier, faster, and offers excellent exposure. As this is the most frequent presentation of dumbbell tumors, the Grillo approach should be considered the standard operation.

When, however, more complex lesions are encountered, a wider exposure is required, and the double approach proposed by Akwari and associates is, in our opinion, preferable. If the tumor involves more than one foramen and three or more laminectomies are required, extended mobilization of paravertebral muscles is mandatory, especially in the presence of bony infiltration or intradural involvement. The spinal lesion should be managed first by the neurosurgeon, who chooses the site and extension of the incision. After laminectomy has been performed and the tumor has been resected up to the posterior arch of the ribs, thoracotomy is carried out. Spinal injury due to coagulation and bleeding in the neural canal is thus avoided, and the intrathoracic tumor can be easily resected.

In conclusion, we think that MRI should always be used in the work-up of patients with neurogenic tumors of the posterior mediastinum to rule out intraspinal involvement and define its extension. We consider the Grillo technique indicated when only one foramen is involved and the intraspinal component is small. Thoracotomy and a longitudinal paravertebral incision are more appropriate when the tumor is large (more than 4 cm) and involves more than one foramen, when the intraspinal extension exceeds 2 to 3 cm requiring multiple laminectomy, and when bony infiltration is present.

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Reoperations on Heart Valve Prostheses: An Analysis of Operative Risks and Late Results

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To evaluate risks and complications of reoperations on heart valve prostheses, we reviewed data on 183 patients who underwent reoperation because of prosthetic valve malfunction. The incremental effect of the redo procedure on hospital mortality and morbidity was studied by comparing primary and reoperative procedures and analyzing a series of possible predisposing factors. Late survival after first and second reoperations was computed, and possible determinants of late mortality were examined. Overall operative mortality was 8.7%; emergency operation (p = 0.0001), previous thromboembolism (p = 0.05), and advanced New York Heart Association functional class (p = 0.031) were the independent determinants. In a series of 1,355 patients having primary or secondary isolated valve replacement, the redo procedure was a significant risk factor in the univariate analysis (p = 0.025) but not in the multivariate analysis except for the subset of patients having mitral valve replacement (p = 0.052). The postoperative course was quite complicated, as evidenced by the long mean stay in the intensive care unit (mean stay, 3.8 days; longer than 2 days for 26% of the survivors). Nevertheless, postoperative complications were not significantly greater after a redo procedure than after a primary operation. Actuarial survival at 7 years was 57.3% ± 8%. A comparison with a nonhomogeneous series from our institution did not demonstrate significant differences. In the subset of 16 patients having a second reoperation, late survival was 37.8% ± 16% at 2 years. Advanced New York Heart Association class (p = 0.0001), double prosthetic valve dysfunction (p = 0.003), and any indication other than primary tissue failure (p = 0.06) were determinants of late mortality. The surgical risk of reoperation on heart valve prostheses is higher than that of the primary operation, but there is no conclusive evidence that the difference is statistically significant. Life expectancy may be reduced, but late survival can be improved if conditions leading to myocardial damage are prevented, ie, reoperation is prompt when necessary.

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Reoperations on heart valve prostheses have become more frequent as use of replacement procedures has become more widespread [1]. Valve reoperations are undertaken for several causes, but one of the most common is structural failure of the prosthesis. This event, although rare for mechanical prostheses, has to be considered part of the natural evolution of bioprostheses, and will further increase the need for valve reoperations in the future. Therefore, we reviewed our data on valve reoperations to add to the knowledge on the risks and complications of these procedures. Also, we believe that this information is helpful in making the decision whether to choose a biological or a mechanical valve at the first intervention.

Material and Methods

Patient Population

The 183 consecutive patients who underwent valve replacement without associated procedures from January 1979 to December 1987 at the University of Torino form the basis of this report. There were 111 male and 72 female patients. Age ranged from 15 to 74 years and averaged 49 ± 14 years (± standard deviation). One hundred thirty-one patients had biological prostheses and 52, mechanical prostheses. The position of the valves was as follows: aortic in 73 patients (40 biological and 33 mechanical), mitral in 91 (73 biological and 18 mechanical), and double, ie, mitral and aortic or mitral and tricuspid, in 19 (18 biological and one mechanical). The form of hemodynamic dysfunction most often encountered was regurgitation, due either to prosthetic valve failure or paravalvular leak. Regurgitation was present in 131 patients, stenosis was found in 22, and both were present in 16. Fourteen patients had other forms of dysfunction.

Definitions

Prosthetic valve endocarditis was suspected when at least two of the following signs occurred: long-lasting intermittent fever, septic embolism, or positive blood cultures. The diagnosis had to be confirmed by histological examination, cultures of the prosthetic valve specimen, or both [2, 3].

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Table 1. Operative Findings and Surgical Treatment

Diagnosis	No. of Patients	Position			Emergency		Duration of Implantation (mo)	Operation		
		Aortic	Mitral	Double	No. of Patients	%		PVR	R	D
Paravalvular leak	56	28	26	2	15	26.8	30 ± 42.2	49	7	0
Endocarditis	41	26^a	13	2	23	56.1^b	27 ± 24.8	41	0	0
Primary tissue failure	64	13	39^b	12	11	17.2	82 ± 23.7	64	0	0
Thrombosis	22	5	14	3	6	27.3	65 ± 40.9	19	0	3

Correlations between diagnosis and both valve position and emergency status have been calculated. Significance: ^{a} $p < 0.002$; ^{b} $p < 0.001$.

D = declotting; PVR = prosthetic valve replacement; R = resuture.

Primary tissue failure was recognized when bioprostheses showed tears or calcifications or a combination of these and there had been no clinical or laboratory evidence of infection [4, 5].

Paravalvular leak included only cases of sterile dehiscence without clinical or cultural signs of endocarditis.

Thromboembolism was assumed when there was either preoperative (echocardiographic) or intraoperative demonstration of prosthetic valve thrombosis, or clinical history of recurrent embolism.

Hospital mortality included all deaths occurring before discharge or within 30 days after operation.

Early morbidity included one or more of the following complications: excessive postoperative blood loss (more than 1,000 mL in 24 hours), chest reexploration, low-output syndrome, ventricular arrhythmias, prolonged mechanical ventilation, renal failure, early thromboembolism, and exceedingly long stay in the intensive care unit. The last has been recognized as a reflection of postoperative morbidity; treatment in the intensive care unit lasting more than 2 days was closely correlated with a complicated postoperative course.

Indications for Operation

The most common condition leading to reoperation was primary tissue failure of a bioprosthesis. It occurred in 64 patients (35% of the total population), 12 of whom had two failing prostheses (Table 1). Other indications were paravalvular leak (56 patients), endocarditis (41), and thrombosis or thromboembolism (22).

Surgical Procedures

All operations were performed with moderate general hypothermia (30°C), pericardial cooling, and cardioplegic arrest with injection in the aortic root of a single dose of St. Thomas' solution. The prostheses were replaced in 173 patients, resutured in 7, and declotted in 3. An emergency operation was performed in 55 patients because of severe congestive heart failure and was required mainly when prosthetic valve endocarditis ($p < 0.001$) was the cause of the malfunctioning (see Table 1).

Comparison With Primary Procedures

To assess the risks of reoperation for prosthetic valve malfunction, we retrieved data on primary isolated valve replacements performed in our institution. Because a complete database was available only for patients operated on from 1979 to 1984, we made comparisons between a population of 1,272 patients who underwent a primary procedure in those years and the 83 patients who underwent valve reoperation in the same period. These 83 patients are among the 183 patients forming the basis of this study.

Follow-up

Total follow-up covers 384.6 years and 95% of hospital survivors. It averages 25 months per patient.

Statistical Analysis

Data were evaluated by computing means and percentages, ± standard deviations, and by t test, χ^2 test, and Fisher's exact test when appropriate. Risk factors of hospital mortality were calculated by univariate analysis and then by introducing variables with a p value of 0.20 or less into a stepwise logistic regression analysis [6]. This analysis was performed both to determine the risk factors in the group of 183 patients having reoperation and to determine whether reoperation had an incremental effect on mortality in the group of patients having isolated valve replacement within a given time frame (83 with a reoperation and 1,272 with a primary procedure). Actuarial rates were calculated according to the method of Cutler and Ederer [7, 8]. Risk factors of late mortality were first evaluated with the tests of Mantel and Breslow and then, if a p value of 0.20 or less was found, with the Cox proportional hazards model [9]. All analyses were performed with the BMDP statistical package.

Results

Malfunction-Related Factors

The type of malfunction was significantly related to the position of the prosthesis. Endocarditis more often affected aortic prostheses ($p = 0.002$), and primary tissue failure more commonly occurred with mitral prostheses ($p = 0.001$). Only paravalvular leaks were equally distributed among the two positions (see Table 1).

Time Intervals

The duration of implantation after the first operation was longest when malfunction was due to primary tissue

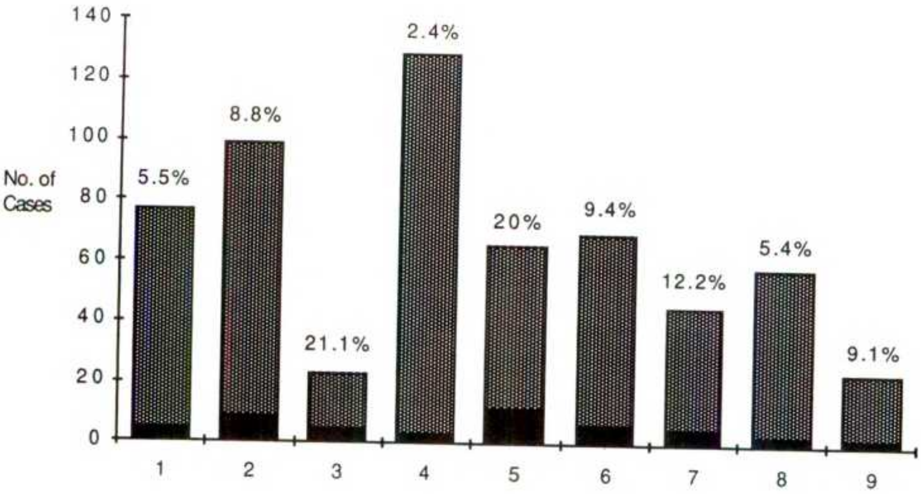

Fig 1. Hospital mortality (%) according to valve position (1 = aortic; 2 = mitral; 3 = double), operative priority (4 = elective; 5 = emergency), and surgical indication (6 = primary tissue failure; 7 = endocarditis; 8 = paravalvular leak; 9 = thromboembolism). There were no significant differences between the different valve positions (p = 0.105) or the indications for operation (p = 0.691). Hospital mortality for emergency procedures was significantly greater than that for elective procedures (p = 0.0001).

failure (mean duration, 82 ± 23.7 months). The shortest interval was that between the first operation and malfunction due to endocarditis (mean duration, 27 ± 24.8 months); in 12 patients, this interval was less than 6 months (see Table 1).

Hospital Mortality

Overall hospital mortality was 8.7% (16/183). The most common cause of death was a low-output syndrome, either acute (6 patients) or chronic (6 patients). Brain abscess, neurological complications, myocardial infarction, and exsanguination at repeat sternotomy accounted for one death each. Emergency reoperation (20%) (p = 0.0001), double-valve re-replacement (mitral and aortic or mitral and tricuspid) (21%) (p = 0.105), and reoperation for early (within 6 months) prosthetic valve endocarditis (16.7%) (p = 0.269) carried the highest mortality (Fig 1). Reoperations necessitated by endocarditis, both early and late, had a hospital mortality of 12.2% (p = 0.36); those secondary to sterile paravalvular leak had the lowest mortality (5.36%) (p = 0.28).

Among risk factors (Appendix 1) evaluated for possible significance in the multivariate analysis, emergency operation (p = 0.0001), advanced New York Heart Association functional (NYHA) class (p = 0.031), and previous thromboembolism (p = 0.05) proved to be independent determinants. Double-valve replacement, age, and female sex had p values of 0.146, 0.205, and 0.268, respectively.

Risk of Hospital Mortality for Primary Versus Reoperative Procedures

In the group of 1,355 patients operated on from 1979 to 1984, hospital mortality was 9.6% for the 83 patients having reoperation compared with 4.3% for the 1,272 patients undergoing primary procedures (p = 0.025). To better assess the predictive value of a redo operation on operative mortality, we performed univariate and multivariate analysis of several possible predisposing factors (Table 2). For the total population, the heavier risk factors in the univariate analysis appeared to be NYHA functional class IV (p = 0.00001), emergency procedure (p = 0.00004), ischemic time (p = 0.0007), associated tricuspid stenosis and regurgitation (p = 0.0045), age (p = 0.0045), age greater than 65 years (p = 0.01), double-valve replacement (p = 0.0152), and reoperative procedure (p = 0.025). In the multivariate analysis, redo procedure could not be demonstrated to influence operative survival (see Table 2).

We also divided the population by operative procedure—mitral valve replacement, aortic valve replacement, and double-valve replacement—to perform the same analysis in each subset (Table 3). A redo procedure appeared to affect early survival only in the subset having mitral valve replacement, as this factor was significant (p = 0.052) also in the logistic regression analysis.

Table 2. Risk Factors for Operative Mortality in 1,355 Patients Having Primary and Redo Procedures

Variable	p Value	
	Univariate	Multivariate[a]
NYHA Class IV	0.00001	0.006
Emergency operation	0.00004	0.0001
Aortic cross-clamp time	0.0007	0.0001
Tricuspid stenosis and regurgitation	0.0045	0.009
Age	0.0045	0.001
Age >65 years	0.01	. . .
Multiple-valve replacement	0.0152	. . .
Reoperation	0.025	. . .
Tricuspid regurgitation	0.055	. . .

[a] Variables showing a p value of greater than 0.2 in the univariate analysis were not entered into the logistic regression analysis: year of operation, sex, etiology, minor coronary artery disease, endocarditis, diabetes, previous noncardiac operation, valve malfunction (stenosis or regurgitation), renal insufficiency, pulmonary artery pressure, ejection fraction, left ventricular end-diastolic pressure, surgical procedure on tricuspid valve, and type of operation (re-replacement, resuture, or declotting). The p values greater than 0.05 in the multivariate analysis are not reported.

NYHA = New York Heart Association.

Table 3. Risk Factors for Operative Mortality for Valve Replacement in Primary and Redo Procedures

	MVR		AVR		DVR	
	p Value		*p* Value		*p* Value	
Variable	Univariate	Multivariate[a]	Univariate	Multivariate[b]	Univariate	Multivariate[b]
NYHA class IV	0.005	0.007	. . .	. . .	<0.00001	. . .
Reoperation	0.04	0.052	. . .	. . .	. . .	. . .
Emergency operation	0.039	. . .	0.0001	. . .	<0.00001	. . .
Age	0.068	0.066	. . .	. . .	. . .	. . .
Age >65 years	. . .	. . .	. . .	. . .	0.058	. . .
Ejection fraction < 0.35	. . .	. . .	0.037	. . .	. . .	. . .
Endocarditis	. . .	. . .	. . .	. . .	0.0083	. . .

[a] The *p* values greater than 0.05 in the multivariate analysis are not reported. [b] Multivariate analysis was not performed, as reoperation was not significant in the univariate analysis.

AVR = aortic valve replacement; DVR = double-valve replacement; MVR = mitral valve replacement; NYHA = New York Heart Association.

Early Morbidity

Catastrophic hemorrhage at repeat sternotomy occurred in only 1 patient, who eventually died of neurological sequelae. Other complications are listed in Table 4. Among these, prolonged mechanical ventilation (more than 18 hours) was the most common (37%). Moreover, the long stay in the intensive care unit (average stay, 3.8 days; longer than 2 days in 25.7% of the survivors) was indicative of the relatively high incidence of a complicated postoperative course.

Early Morbidity for Primary Versus Reoperative Procedures

The postoperative course of the 83 patients undergoing reoperation was not significantly more complicated than that of the 1,272 patients undergoing primary procedures during the same period, 1979 to 1984 (Table 5). A longer stay in the intensive care unit (more than 2 days) occurred more often after a reoperation (27.5% versus 21.5%), but the difference was not significant ($p = 0.23$). Likewise, excessive postoperative bleeding (more than 1,000 mL in the first 24 postoperative hours) occurred more often after a redo procedure (14.5% versus 12.6%) and sternal infection occurred slightly more often after a redo procedure (1.3% versus 1.1%), but the differences were not significant ($p = 0.64$ and $p = 0.93$, respectively). Although patients having reoperation received more blood transfusions (1,126 mL versus 796 mL; $p = 0.038$), they were not reexplored for excessive bleeding more often than patients at first operation (8% versus 9.8%; $p = 0.6$).

Late Survival

Actuarial survival of all 183 patients at 5 years (29 patients at risk) was 66.9% ± 5% and at 7 years, 57.3% ± 8% (Fig 2). Actuarial survival at 7 years was 42% ± 14% for patients having aortic valve replacement, 72% ± 9% for patients having mitral valve replacement, and 48% ± 16% for patients having double-valve replacement (Fig 3).

We calculated the operation-free survival to compare the subset of patients undergoing the first reoperation with the subset undergoing the second reoperation. In the reoperation-free rates, those patients who underwent a second reoperation are removed from the first-reoperation curve at the time of the second intervention. The operation-free survival at 2 years for patients having one

Table 4. Postoperative Complications[a]

Complication	No. of Patients[b]
Prolonged mechanical ventilation	67 (36.6)
Thromboembolism	9 (4.9)
Low-output syndrome	21 (11.5)
Ventricular arrhythmias	29 (15.8)
Renal failure	3 (1.6)
Reexploration	16 (8.7)
Bleeding (mL)	862 ± 778
ICU stay (d)	3.78 ± 6.7
ICU stay > 2 days	47 (25.7)

[a] Numbers in parentheses are percentages. [b] Data are shown as number of patients unless otherwise indicated.

ICU = intensive care unit.

Table 5. Postoperative Morbidity

Variable	First Operation	Reoperation	*p* Value
ICU ≥3 days	21.5% (254/1,183)	27.5% (19/69)	0.23
Excessive postoperative bleeding	12.6% (149/1,184)	14.5% (10/69)	0.64
Mediastinitis	1.1% (13/1,212)	1.3% (1/75)	0.90
Postoperative transfusions	796 ± 19 mL	1,126 ± 154 mL	0.038
Reexploration for excessive bleeding	9.8% (119/1,212)	8% (6/75)	0.60

Patients with missing values in the database have been excluded.

ICU = intensive care unit.

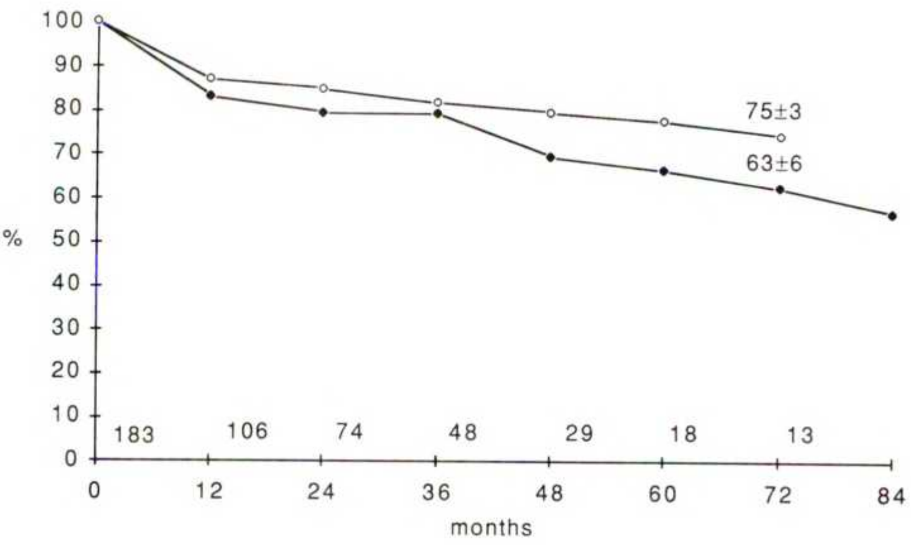

Fig 2. Actuarial survival of patients having reoperation because of prosthetic valve malfunction (—◆—). At 5 years, survival was 66.9% ± 5% and at 7 years, 57.3% ± 8%. The number of patients at risk is indicated for each interval. The upper curve (—◇—) indicates the actuarial survival of a noncontemporary population of patients undergoing primary procedures in our institution. There is no significant difference between the two curves (p = 0.093 at 72 months).

reoperation was 74.9% ± 3.6% and that of the 16 patients reoperated on twice, 37.8% ± 16% (Fig 4).

Determinants of late mortality according to the Cox model were the following: advanced NYHA functional class ($p = 0.0001$) and double prosthetic valve dysfunction ($p = 0.003$). Any indication other than primary tissue failure ($p = 0.061$), second reoperation ($p = 0.124$), and ventricular arrhythmias ($p = 0.171$) were close to significance.

Comment

Reoperations on valve prostheses present several problems: the possible influence of specific factors (eg, the type of prosthetic valve malfunction) and the possible incremental effect of the redo procedure on hospital mortality; enhanced perioperative morbidity; reduced life expectancy after the first and after the second reintervention; and possible factors related to reduced late survival. The high mortality often associated with reoperations is partly due to the severity of a frequent cause of the redo procedure: prosthetic valve endocarditis. Indeed, endocarditis is reported to increase operative mortality significantly [10–15]. In our series, endocarditis led to the highest mortality rate among the causes of prosthetic valve malfunction (see Fig 1). Nevertheless, in the multivariate analysis, it did not appear to be an independent significant risk factor.

The treatment of another common cause of redo procedures, primary tissue failure, has also not met with much success in our experience: hospital mortality for re-replacement after primary tissue failure was 9.4% (see Fig 1). This result could have been related to the fact that the subset of 64 patients with primary tissue failure included many at high risk: 11 (17.2%) required an emergency operation, 9 others (14.1%) were in NYHA class IV, and 12 (18.8%) underwent double re-replacement because of failure of both prostheses. Nevertheless, like prosthetic valve endocarditis, primary tissue failure did not appear to be an independent risk factor in the multivariate

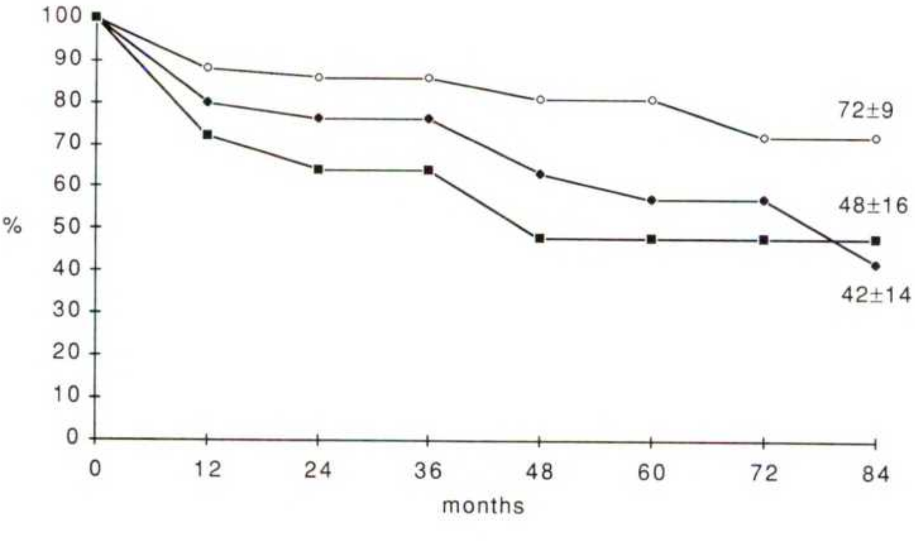

Fig 3. Actuarial survival of patients undergoing reoperation because of malfunction of aortic (—◆—) or mitral (—◇—) valve prostheses or two (—■—) valve prostheses (mitral and aortic or mitral and tricuspid). Survival after reoperations on aortic prostheses and two failing prostheses was particularly low.

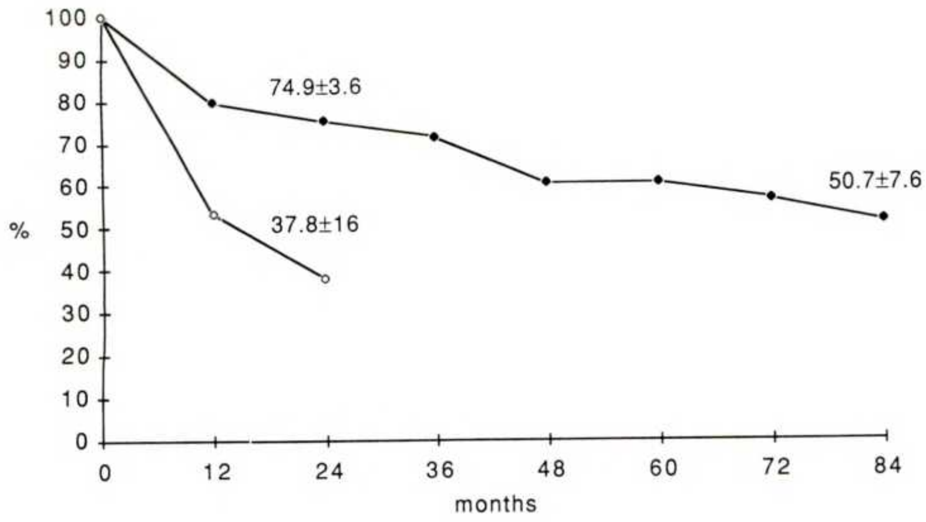

Fig 4. Operation-free survival after the first reoperation (—◆—) and after the second reoperation (—◇—). Reoperation-free rates remove patients who underwent a second reoperation from the curve of first reoperation. After 2 years, survival of patients having two reoperations, 37.8% ± 16%, compares unfavorably with that of patients having but one reoperation, 74.8% ± 3.6% (p = 0.024).

analysis. Indeed, our multivariate analysis demonstrated that only emergency operation, advanced NYHA class, and previous thromboembolism were significant predictors of hospital mortality. Our results confirm previously reported data as far as emergency operation (11–14, 16, 17] and advanced NYHA class [10, 14, 16, 18] are concerned.

The main question about reoperations is whether they have an incremental effect on operative mortality compared with primary procedures. The reported data are controversial [10–13, 16, 18, 19], but in recent years, there has been a definite trend toward neutralization of the risk, particularly since the adoption of routine cardioplegic arrest [12]. In our experience, reoperations could be performed with an acceptable hospital mortality, although higher than that of primary valve replacements. However, we have not been able to demonstrate the possible incremental effect of reoperative procedures. Multivariate analysis of predictors of hospital mortality after prosthetic valve replacement, both primary and secondary, has emphasized the role of emergency procedure and functional class, but has not indicated redo procedure to be a significant determinant (see Table 2), except for the subset of patients having mitral valve replacement (see Table 3). On the other hand, our series included a relatively large number of reoperations due to paravalvular leak (31%), which are considered a low-risk category [13]. This could have made us underestimate the actual influence of the redo procedure.

From a practical perspective, what can we do to further reduce surgical mortality when treating malfunctioning prostheses? Patients should be operated on as soon as possible to avoid deterioration of the clinical status. A failing bioprosthesis should be considered suspect, as a partially safe condition can soon become an unduly risky situation. We suggest reoperation for all failing bioprostheses with proven tears or calcifications, even though the patients are asymptomatic or only mildly symptomatic.

Perioperative morbidity can be detrimental, and recovery from reoperations is likely to be very complicated. The occurrence of postoperative complications was frequent in our series (see Table 4). Nevertheless, in the subset of patients having reoperation compared with those having primary procedures, there were no significant differences with the exception of postoperative blood transfusions (see Table 5).

Another possible drawback is the quality of late results. Blackstone and Kirklin [1] reported a shorter life expectancy for patients undergoing reoperation. In our patient population, the actuarial survival at 7 years was only 57% (and the survival of patients having aortic valve replacement or double-valve replacement was particularly low, as indicated in Figure 3), but in a previous report from our institution [20], the survival rate at 7 years in a series of 990 consecutive patients having primary valve replacement was 75%. Our results are not conclusive: the present data compare unfavorably with the earlier data, but there is no significant difference between the two curves (see Fig 2).

The evaluation of risk factors of late mortality indicates that patient-related variables have the greatest weight. When the Cox model was applied to our data, it demonstrated that advanced heart disease (advanced NYHA class) is the main independent predictor of late mortality. This implies that myocardial damage, more common with long-term diseases, such as are usually associated with prosthetic valve malfunction, is the chief factor jeopardizing late survival [21]. Other results of our analysis tend to reinforce this conclusion; re-replacement of two failing prostheses and postoperative ventricular arrhythmias, both suggesting strain on the left ventricle, have a p value of 0.003 and 0.171, respectively. Patients undergoing reoperation because of primary tissue failure had the better life expectancy ($p = 0.061$) compared with those having reoperation because of endocarditis, paravalvular leak, or thrombosis. This might be due to the fact that primary tissue failure affects only the prosthesis itself, provided reoperation is judiciously prompt. On the other hand, endocarditis, paravalvular leak, and thrombosis are often related to systemic pathological or anatomical causes difficult to neutralize or eradicate.

A second reoperation, which suggests a longer disease interval, can further compromise late survival, a hypothesis already put forth by Blackstone and Kirklin [1]. That this is true is quite clear from the comparison of first and second reoperations shown in Figure 4, where operation-free survival rates are presented.

Conclusions

We believe that the surgical risk of reoperation on heart valve prostheses does not dramatically differ from that of primary operations, particularly if the intervention is not unduly delayed. Late survival can be improved if conditions leading to myocardial damage are prevented, ie, reoperations are performed when necessary. Therefore, we believe that bioprostheses can be safely chosen at primary valve replacement in many subsets of patients [22], as the risk of re-replacement seems to be lower than the dangers of thromboembolism and anticoagulation that occur with mechanical devices [23, 24]. Patients whose condition is severely compromised should be excluded; a second operation could be detrimental because the results that can be achieved in such patients are very poor.

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Appendix 1. Risk Factors for Hospital Mortality

Variable	*p* Value
Age	0.16
Sex	0.05
Previous emboli	0.004
Hemodynamic dysfunction	0.47
Time elapsed from implantation	0.15
Aortic cross-clamp time	0.86
Blood loss	0.20
NYHA class (mean)	0.006
Valve position	0.10
Diagnosis (leak, endocarditis, primary tissue failure, or thrombosis)	0.69
Type of prosthesis	0.31
Emergency operation	0.00001
Type of operation (re-replacement or other)	0.22
Transfusions	0.03

Complement Activation and Lung Permeability During Cardiopulmonary Bypass

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Pulmonary dysfunction after cardiopulmonary bypass has been attributed to the damaging effects of complement activation on the lung. To further explore this phenomenon, we measured plasma levels of activated complement components (radioimmunoassay), assessed neutrophil *n*-formyl-methionyl-leucyl-phenylalanine (FMLP) receptor status (radioligand saturation binding assay), and quantified pulmonary epithelial permeability as radioaerosol lung clearance of technetium 99m–labeled diethylenetriamine pentaacetic acid in a series of 8 patients undergoing cardiopulmonary bypass. Significant elevations of plasma C3adesArg, C4adesArg, and C5adesArg levels were seen just after CPB, indicating activation of both the classic and alternate complement pathways. Neutrophil activation was evident as increased expression of neutrophil FMLP surface receptors after bypass. Despite the presence of complement and neutrophil activation, increased pulmonary epithelial permeability was not seen. These data support the hypothesis that complement and neutrophil activation during cardiopulmonary bypass is not associated with acute lung injury, at least not pulmonary epithelial injury. One can therefore infer that increased pulmonary epithelial permeability in patients at high risk for and experiencing sepsis-induced and trauma-induced adult respiratory distress syndrome may be due to factors other than complement and neutrophil activation.

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As a result of the interaction of blood with components of extracorporeal circulation, cardiopulmonary bypass (CPB) is associated with intense complement activation [1, 2]. Complement activation and its resultant neutrophil (PMN) activation is, in turn, believed to contribute to the pulmonary dysfunction associated with CPB. The clinical manifestations of CPB-associated pulmonary dysfunction include atelectasis, pulmonary edema, and, rarely, acute lung injury seen as adult respiratory distress syndrome (ARDS). Complement activation, with PMNs, platelets, or both as mediators and the pulmonary alveolar-capillary membrane as target, is believed to be one pathophysiological mechanism accounting for these phenomena [3, 4].

We [5] have shown that subclinical abnormalities in pulmonary epithelial permeability (PEP), detected as increased radioaerosol lung clearance, precede the clinical manifestations of ARDS in patients experiencing sepsis and multiple trauma. In addition, many patients experience subclinical acute lung injury that resolves without apparent clinical sequelae. Because the pathophysiology of CPB-associated pulmonary dysfunction may be similar to that seen in sepsis-related and trauma-related ARDS, we studied the relationship between complement and PMN activation during CPB and PEP.

Material and Methods

Eight nonsmoking adults undergoing CPB for elective coronary artery bypass grafting were studied. Seven of the 8 patients were men, and the mean age of the group was 62.4 ± 3.4 years. All patients were operated on by the same surgeon (W.W.B.). The CPB technique was identical for all procedures and consisted of a roller-pump with bubble oxygenator, asanguineous prime with mannitol, moderate hypothermia, and routine heparinization and protamine sulfate reversal. Mean time on CPB for the group was 113 ± 3 minutes, and cross-clamp time averaged 58 ± 7 minutes. The study was approved by the Human Research Committee of the University of Cincinnati College of Medicine (May 28, 1986), and patients provided informed consent before participation.

Complement Assays

Arterial blood samples were collected in plastic syringes and immediately transferred into Vacutainer tubes (Benton Dickinson Inc, Rutherford, NJ) containing EDTA (ethylenediaminetetraacetic acid). Samples were placed on ice and centrifuged (1,200 *g* for 10 minutes) at 4°C, and the harvested plasma was stored in polypropylene tubes at −80°C until assay. C3adesArg, C4adesArg, and C5adesArg, the metabolites of the respective activated complement components, were measured by double-antibody radioimmunoassay using commercially available kits (Amersham Corp, Chicago, IL) [6]. Values were not corrected for hemodilution because the in vivo effect of activated complement components is a function of the

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true plasma concentration and hemodilution correction makes comparisons with other clinical settings difficult.

Neutrophil FMLP Receptor Status

Neutrophils were harvested from blood according to the technique of Boyum [7] with several modifications as previously described [8]. Briefly, this involved dextran sedimentation, Ficoll–sodium diatrizoate density gradient centrifugation, and hypotonic lysis of contaminating red blood cells. The entire isolation was carried out at 4°C to prevent temperature-related receptor mobilization and inadvertent cellular activation [8]. The *n*-formyl-methionyl-leucyl-phenylalanine (FMLP) receptor number and affinity were determined by a radioligand (^{3}H-FMLP; New England Nuclear, Boston, MA) saturation binding assay. Varying concentrations of radioligand were added to 2×10^6 PMNs in the absence or presence of 10^{-4} mol/L unlabeled FMLP for the determination of total and nonspecific binding, respectively. The FMLP receptor number and affinity were determined from Scatchard analysis of plots of specific binding (total binding minus nonspecific binding) versus the bound to free ratio. This assay has been characterized in our laboratory [8].

Pulmonary Epithelial Permeability

Pulmonary epithelial permeability was determined as the radioaerosol lung clearance of technetium 99m–labeled diethylenetriamine pentaacetic acid (^{99m}Tc-DTPA). This is an inert hydrophilic radiotracer that crosses the pulmonary alveolar-capillary membrane and provides a measure of PEP. After the administration of a ^{99m}Tc-DTPA microaerosol through a mouthpiece, the pulmonary clearance, from lung to blood, was monitored with a portable gamma camera. Computer interfacing allowed generation of time–activity curves for each lung. The clearance from each lung was calculated from the slope of time–activity curves generated from the first 7 minutes of isotope washout and expressed as percent decline per minute. Clearance from each lung was averaged to determine the final value for each patient. A detailed review and validation of this technique have been published [9]. Of particular note are the lack of dependence of PEP on pulmonary blood flow and hence regional perfusion, the homogeneous distribution of the radiotracer in all patients studied, the fact that scanning patients anteriorly or posteriorly in the supine position does not affect clearance, and the lack of an effect of general anesthesia with surgical procedures or thoracotomy on PEP.

Experimental Protocol

Plasma samples for complement assays were collected during the following time points: prebypass (immediately before institution of CPB), on bypass (30 minutes into CPB), postbypass (30 minutes after cessation of CPB), postoperative (4 hours postoperatively), postoperative day 1, and postoperative day 3 to 5. Determination of PMN FMLP receptor status and lung scanning were performed at the times indicated. Prebypass lung scans were obtained the day before operation.

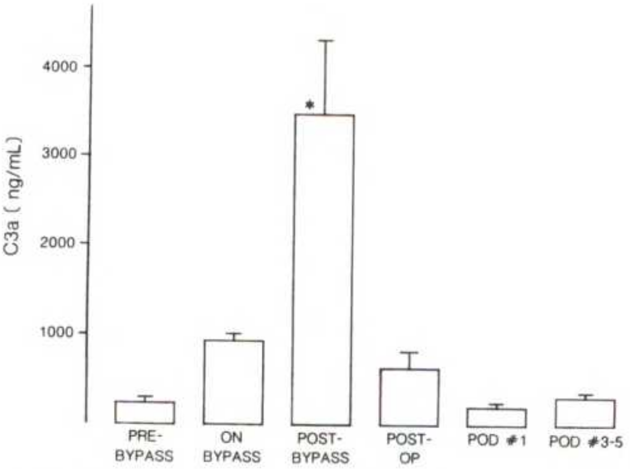

*Fig 1. Plasma levels of C3adesArg during and after cardiopulmonary bypass (n = 8). (POD = postoperative day; * = p < 0.05 versus prebypass level by repeated-measures analysis of variance.)*

Statistical Analysis

Data are expressed as the mean ± the standard error of the mean. A repeated-measures analysis of variance followed by Duncan's multiple range test was used to test for significant changes over the time course of these experiments. Correlation analysis and Student's paired *t* test were used as indicated.

Results

Figures 1 through 3 demonstrate the pattern of complement activation during and after CPB. Plasma levels of activated complement components rose during bypass and peaked during the postbypass period. This was most striking for C3adesArg, which underwent a nearly 15-fold increase during this period. C4adesArg levels increased 11-fold, and C5adesArg levels increased 1.6-fold ($p < 0.05$

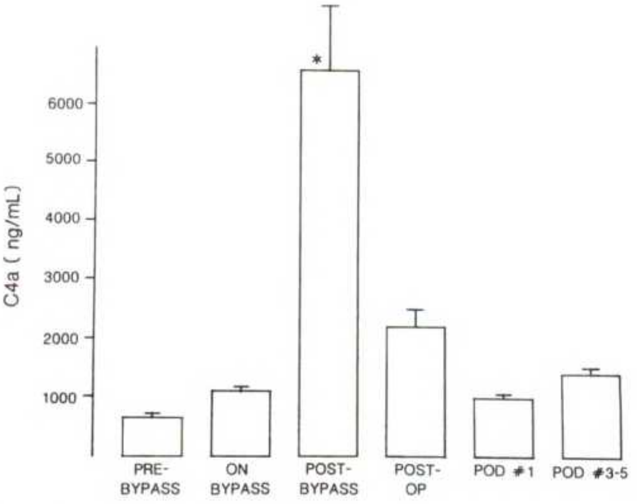

*Fig 2. Plasma levels of C4adesArg during and after cardiopulmonary bypass (n = 8). (POD = postoperative day; * = p < 0.05 versus prebypass level by repeated-measures analysis of variance.)*

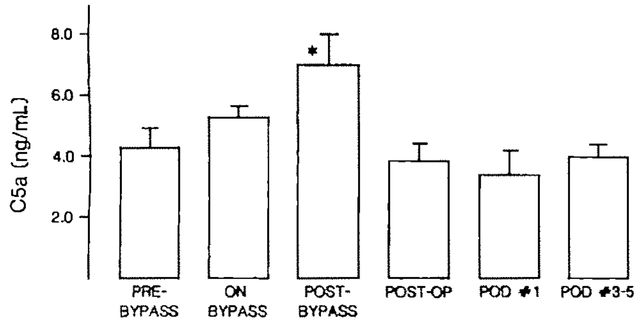

*Fig 3. Plasma levels of C5adesArg during and after cardiopulmonary bypass (n = 8). (POD = postoperative day; * = p < 0.05 versus prebypass level by repeated-measures analysis of variance.)*

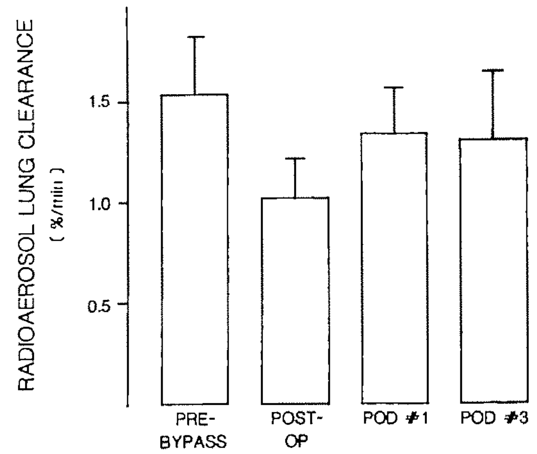

Fig 4. Radioaerosol lung clearance before and after cardiopulmonary bypass (n = 7 or 8). Adult respiratory distress syndrome developed in 1 patient (data not included) in the postoperative period, and clearance increased to 4.0%/min. No significant changes were found by repeated-measures analysis of variance. (POD = postoperative day.)

versus prebypass values). Levels remained elevated during the postoperative period (except for C5adesArg) and returned to baseline by the first postoperative day.

Cardiopulmonary bypass was associated with an increased expression of PMN surface receptors for the chemotactic peptide FMLP. Although there was a 48% increase in receptor number, the affinity of the expressed receptor was unchanged (Table 1). The single-affinity receptor model, as opposed to a double-affinity system, was preserved in all instances (data not shown).

Radioaerosol lung clearance, measured with the ^{99m}Tc-DTPA probe, was unchanged in the immediate postoperative period and remained unchanged on postoperative days 1 and 3 (Fig 4). Of note, ARDS developed in 1 patient on postoperative day 1, presumably secondary to subsequent hemorrhagic shock, multiple blood transfusions, and the need for additional CPB. Pulmonary epithelial permeability rose to 4.0%/min for this patient.

Except for patient 2 (Table 2), all patients were extubated on postoperative day 1 and were free from any major pulmonary complications.

A series of correlation analyses were performed to determine whether any relationship existed between measurements of pulmonary function and degrees of complement and PMN activation. These analyses were carried out using individual patient data at the postoperative time point of the study. This time point was chosen because lung injury was expected to be maximal at this time and because patients had full instrumentation and were carefully monitored (arterial lines, pulmonary artery catheters, and endotracheal ventilatory support), thus allowing accurate determinations of alveolar–arterial oxygen tension gradients and intrapulmonary shunts. No significant correlations were found between the alveolar–arterial oxygen tension gradient or PEP as determined by radioaerosol lung scan and serum levels of the activated complement components (C3adesArg, C4adesArg, and C5adesArg) or PMN FMLP receptor expression (Pearson correlation coefficients ranged from −0.40 to 0.42; *p* values ranged from 0.35 to 0.97; n = 6 to 7). A significant correlation did exist, however, between PEP and the alveolar–arterial oxygen tension gradients at the postoperative time point. The data for this correlation are shown in Table 2.

Table 1. Effect of Cardiopulmonary Bypass on Neutrophil n-Formyl-Methionyl-Leucyl-Phenylalanine Receptor Expression[a]

Time	Receptors per Neutrophil	Receptor Affinity (nmol/L)
Prebypass	4,031 ± 501	22.4 ± 5.5
Postbypass	5,986 ± 894[b]	29.5 ± 4.9

[a] Data are shown as the mean ± the standard error of the mean. [b] Significance: $p < 0.05$ versus prebypass figure (Student's paired *t* test); n = 8.

Table 2. Radioaerosol Lung Clearance and Cardiopulmonary Physiological Measurements Immediately After Cardiopulmonary Bypass[a]

Patient No.	Alveolar–Arterial Oxygen	Intrapulmonary Shunt	Cardiac Output	Pulmonary Epithelial Permeability
1	153	10.4	5.28	0.66
2[b]	. . .	. . .	. . .	. . .
3[c]	262	11.5	3.45	. . .
4	265	22.8	6.04	1.85
5	114	16.7	4.80	0.95
6	190	10.6	4.98	1.01
7	63	6.2	4.53	0.54
8	120	12.0	5.48	1.10

[a] A significant correlation was found between the alveolar-arterial oxygen tension gradient and the radioaerosol lung clearance (Pearson correlation coefficient, 0.84; $p = 0.036$; n = 6). [b] This patient sustained hemorrhagic shock as a result of postoperative bleeding and required further operative intervention. [c] Logistical problems created by the late time of day of this patient's operation precluded a determination of pulmonary epithelial permeability at the 4-hour postoperative time point.

Comment

Complement and PMN activation during sepsis and trauma is often associated with subclinical elevations of PEP that, in many patients, precede the clinical development of ARDS [5, 10]. In this and other settings, complement-mediated PMN activation has received considerable support as an important pathophysiological mechanism in the development of acute lung injury [11–13]. This is particularly true for CPB. Since the work of Kirklin and colleagues [3] and others [1, 2], CPB has been considered a paradigm for the theory of complement-mediated acute lung injury. Our present study confirmed the presence of complement and PMN activation during CPB. This was evident as the generation and accumulation of activated complement components in plasma and the detection of surface receptor mobilization on circulating PMNs. However, despite use of a sensitive measure of PEP, alterations in PEP indicative of clinical and subclinical acute lung injury were not found.

In the present study, intense activation of both the classic (C4adesArg) and the alternate complement pathways was observed during CPB. C5a probably represents the more important activated complement component, as it is a potent effector of PMN function and activation [14]. Detection of elevated C5adesArg levels has traditionally been difficult to document during CPB and in other clinical settings, and evidence of its activation has usually been derived indirectly from PMN function studies, most notably chemotactic deactivation to C5a [6]. Although the rise in C5adesArg levels was hardly as impressive as that of the other complement components, this can be attributed to its binding to high-affinity C5a receptors on the surface of the PMNs [6, 15].

Increased FMLP receptor expression on PMNs after CPB serves as an indicator of PMN activation, particularly enhanced oxidative responses toward stimulation with FMLP [8]. The precise biological importance of the PMN FMLP receptor and FMLP-induced PMN responses remains to be elucidated. However, their potential in vivo importance lies in the fact that FMLP is released by bacteria and degenerating eukaryotic mitochondria at sites of tissue injury and therefore serves as a model for PMN responses toward infectious and inflammatory challenges [16, 17]. Although a correlation between complement activation and increased PMN FMLP expression has been shown in CPB, causality cannot be assumed. Many inflammatory mediators, in addition to C5a, have been shown to increase FMLP receptor expression and enhance FMLP-induced PMN responses in vitro. These include platelet-activating factor, tumor necrosis factor, granulocyte-macrophage colony–stimulating factor, and lipopolysaccharide, and the role of all of these in CPB remains to be elucidated [18–21].

The major finding of this study is that after CPB, serving as a clinical model for perhaps isolated complement activation, evidence of acute lung injury as assessed by radioaerosol lung clearance was not seen. This was manifested as normal PEP, as assessed by ^{99m}Tc-DTPA radioaerosol lung clearance, after CPB, which specifically implies that CPB is not associated with permeability-altering damage to the alveolar component of the pulmonary alveolar-capillary membrane. Because of the rate-limiting role of the pulmonary epithelium in regulating solute flux across the alveolar-capillary membrane, subclinical damage to the capillary endothelium cannot be ruled out [9]. It is possible that the moderately elevated alveolar–arterial oxygen tension gradients and intrapulmonary shunts that were observed postoperatively are a reflection of pulmonary endothelial injury. Although we cannot prove or disprove this, it is more likely that these physiological abnormalities reflect an expected degree of ventilation-perfusion mismatching in a mechanically ventilated patient immediately after CPB. Despite evidence of intense complement activation and associated PMN activation during CPB, pulmonary dysfunction, if present, cannot be attributed to alterations in or damage to the alveolar component of the pulmonary alveolar-capillary membrane.

We have shown that complement and PMN activation during CPB is not associated with the development of acute lung injury. There are other possible explanations for the lack of demonstrable acute lung injury after CPB. First, the duration of complement activation may be too brief. Prolonged complement activation, as seen during sepsis or multiple trauma, may be necessary for pulmonary damage to occur. Second, some component of CPB may protect the alveolar-capillary membrane from injury. In support of this hypothesis, a study [22] from Britian demonstrated increased ^{99m}Tc-DTPA radioaerosol lung clearance after CPB. Although the CPB technique used in that study is quite similar to the technique used in this one, it is possible that a factor not yet identified accounts for the striking difference. Third, it is possible that the radioaerosol clearance technique is not sensitive enough to detect very subtle lung injury. We do not believe this to be the case, however, because in our experience, the technique has been able to detect subclinical abnormalities in PEP in other patient populations and would thus appear to be quite sensitive.

Finally, if we are to believe that perhaps isolated complement and PMN activation, as seen during CPB, is not capable of inducing detectable acute lung injury, the role of these factors in the development of sepsis-induced and trauma-induced ARDS must be reexamined. Indeed, previous data from our laboratory [5] support this theory, namely, that the increased clearance of ^{99m}Tc-DTPA seen in patients at high risk for and experiencing ARDS is due to factors other than complement and PMN activation.

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Complications After Cardiac Operations in Patients With Severe Pulmonary Impairment

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The postoperative courses of 39 patients with severe lung disease (31 with obstructive disease and 8 restrictive) who underwent a cardiac operation were retrospectively reviewed. The stay in the intensive care unit of the study group was 7.9 ± 10.3 days (mean ± standard deviation) compared with 2.4 ± 3.9 days for the control group (100 patients with less impaired pulmonary function) ($p < 0.001$). The study group also had a greater number of valve replacements than did the control group ($p < 0.01$). Patients with obstructive disease had more respiratory complications than did patients with restrictive disease ($p < 0.05$). There were 21 cases of atelectasis. Effusions were noted in 11 patients. Ten patients had bronchospasm. Bronchial secretions were a major problem in 6 patients. Pneumonia developed in 4 patients, and pneumothorax occurred in 3 others. The two in-hospital deaths were not directly related to pulmonary complications. Our findings indicate that (1) patients with severe lung impairment generally do well after a cardiac operation but have more postoperative pulmonary complications than patients with less impairment; (2) patients with restrictive pulmonary disease appear to fare better than those with obstructive disease; (3) pulmonary function tests can alert the clinician to the possible risk of postoperative complications, but they cannot, by themselves, be used to exclude patients from operation; and (4) patients with severe pulmonary impairment facing valve replacement are at greater risk of pulmonary complications than patients having other types of cardiac surgical intervention.

(Ann Thorac Surg 1990;50:602–6)

Most reviews concerning pulmonary complications after cardiac surgical procedures were published in the 1950s and 1960s when surgical techniques were not as well developed as they are today and most patients were operated on for simple congenital or acquired valvar heart disease. Today, most adult patients undergo coronary artery bypass grafting or valve replacement, or both. In addition to improved surgical technique, various forms of ventilatory support and hemodynamic monitoring now in common use were not available two or three decades ago. Advances in pharmacology have also greatly improved the postoperative management of critically ill patients. As a consequence, even patients with serious pulmonary disease are being operated on more frequently.

In spite of all of these improvements, pulmonary function is reduced after open heart operations. Vital capacity, inspiratory capacity, functional residual capacity, total lung capacity, and pulmonary diffusing capacity have been reported to be reduced 2 weeks postoperatively [1]. The values for these variables improved over time but were still significantly decreased from preoperative levels 4 months postoperatively [1].

In view of these changes, one would expect a priori that patients with lung disease are at higher risk for pulmonary complications after cardiac procedures than patients with normal pulmonary function. Several studies [2–4] have shown a higher incidence of pulmonary complications after a variety of thoracic and nonthoracic surgical procedures. No strict pulmonary function guidelines, however, were proposed to help the clinician identify the patients at greater risk.

The following retrospective study was undertaken to determine the postoperative pulmonary complication rate, the morbidity rate, and the mortality rate of cardiac surgical patients with severely impaired preoperative pulmonary function, and to see if conventional pulmonary function tests could be used to identify patients with pulmonary disease severe enough to preclude surgical intervention because of excessive risk for death due to pulmonary complications.

Material and Methods

To assess the postoperative course of patients with severely abnormal pulmonary function, we retrospectively reviewed the hospital records of 39 patients meeting our criteria for severely impaired pulmonary function based on presurgical pulmonary function test data. This study group was drawn from patients who had pulmonary function testing before cardiac operation between 1985 and 1987. Pulmonary function was defined as being severely impaired if four of the seven following criteria were met:

Forced vital capacity (FVC) < 70% (predicted)
Forced expiratory volume in 1 second (FEV_1) < 60% (predicted)

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FEV$_1$/FVC $< 60\%$ (predicted)
Mean forced expiratory flow during middle half of FVC $< 50\%$ (predicted)
Maximal voluntary ventilation $< 50\%$ (predicted)
Arterial carbon dioxide tension > 45 mm Hg
Arterial oxygen tension > 55 mm Hg

The limits of each variable are consistent with published criteria suggesting increased surgical risk [5].

Although total lung capacity was not routinely measured, the patients were classified into those with obstructive or restrictive pulmonary disease on the basis of the FEV$_1$/FVC ratio using published guidelines for pulmonary obstruction [6]. Patients with a ratio of 69% or less were classified as having primarily obstructive disease, and those with a ratio of 70% or greater were classified as having primarily restrictive disease.

Preoperative pulmonary function was correlated with postsurgical recovery on the basis of type of operation performed, length of stay in the intensive care unit (ICU), postsurgical respiratory and nonrespiratory complications, and mortality rate. Respiratory complications were atelectasis, pleural effusion, pneumothorax, bronchospasm, and pneumonia. Nonrespiratory complications were complicated arrhythmias, cardiogenic edema, cardiac tamponade, renal failure, and nonpulmonary infections.

In accord with previous work [4], length of ICU stay was used as an index of the overall complication rate. These data for the study group were compared with similar data for a control group, a single randomly chosen series of 100 patients who had undergone a cardiac operation. Because pulmonary tests in this hospital are performed only in cardiac surgical patients with a previous history of pulmonary disease or those with a high risk for pulmonary impairment (ie, heavy smokers), we assumed that the control group accurately represented a population without pulmonary impairment.

Statistical treatment of nonparametric data was performed using χ^2 analysis. Parametric data were analyzed using analysis of variance and linear regression analysis.

Table 1. Summary of Pulmonary Function Test Results

Variable	Mean	Standard Deviation	Standard Error of Mean	Range
Age (y)	63.8	7.8	1.2	80–39
FVC	61.3	13.6	2.2	90–36
FEV$_1$	49.0	12.0	1.9	80–24
FEV$_1$/FVC	56.0	17.6	2.0	78–40
FEF25–75%	27.3	11.0	1.8	50–58
MVV	38.3	10.8	1.8	60–16
PaO$_2$	59.3	5.3	0.8	72–45
PaCO$_2$	41.7	4.4	0.7	50–31

FEF25–75% = mean forced expiratory flow during middle half of FVC; FEV$_1$ = 1-second forced expiratory volume; FVC = forced vital capacity; MVV = maximal voluntary ventilation; PaCO$_2$ = arterial carbon dioxide tension; PaO$_2$ = arterial oxygen tension.

Results

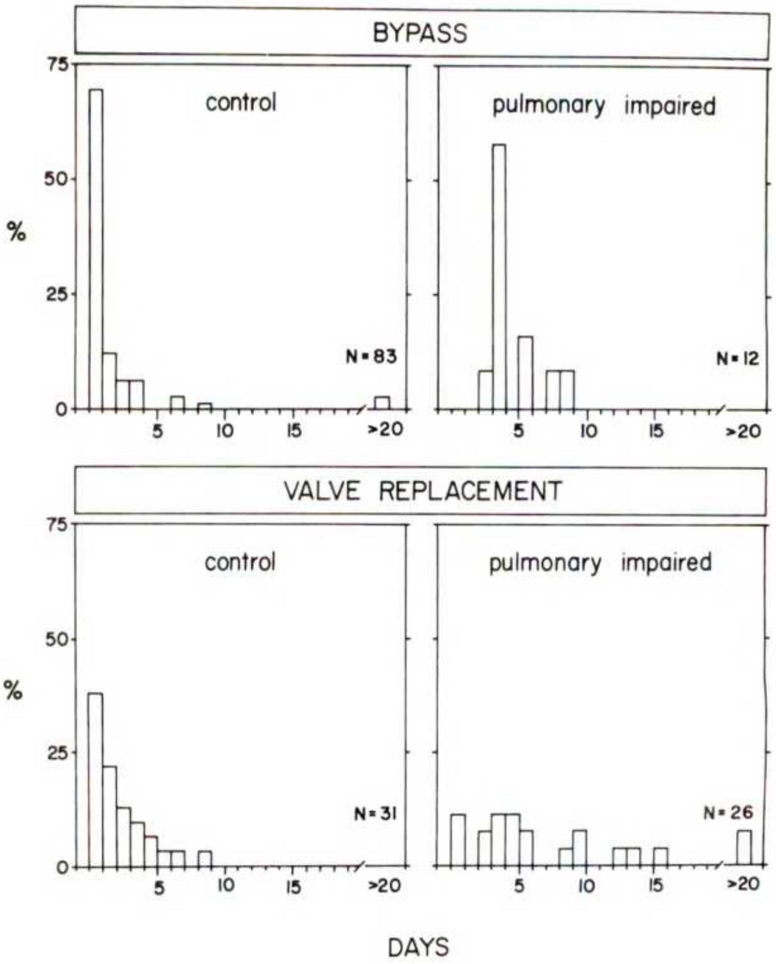
Table 1 summarizes the pulmonary function test results. The surgical procedures in the 39 patients were as follows: coronary artery bypass grafting, 12 patients; coronary bypass with ventricular aneurysm repair, 1 patient; aortic valve replacement, 2; aortic valve replacement with mitral valve commissurotomy, 1; aortic valve replacement with coronary bypass, 3; aortic and mitral valve replacement, 4; mitral valve replacement, 8; mitral valve replacement with coronary bypass, 2; mitral valve replacement with coronary bypass and tricuspid annuloplasty, 1; mitral and tricuspid valve replacement, 4; and ventriculoseptal repair, 1. Compared with the normal distribution of cardiac surgical procedures performed in this institution, this group had a disproportionate number of valve replacements (64% versus 18%; $p < 0.01$). Based on the FEV$_1$/FVC ratio, 31 patients were classified as having primarily obstructive pulmonary disease and 8, restrictive pulmonary disease. Although all patients in this group had some complications, the patients with obstructive pulmonary disease had significantly more respiratory complications in general (50 complications in 31 patients) than patients with restrictive pulmonary disease (7 complications in 8 patients) ($p < 0.05$, χ^2 analysis). There were no significant differences in the rate of nonrespiratory complications between patients with obstructive and those with restrictive disease.

There were 21 cases of atelectasis, making it the most prevalent respiratory complication. There were 11 cases of pleural effusions. Ten patients had episodes of bronchospasm necessitating bronchodilator therapy. All 10 had evidence of obstructive disease on pulmonary function testing, but only 5 had a preoperative history of bronchospasm. Bronchopulmonary secretions were judged a major problem in 6 of the patients with obstructive disease, but only 2 required bronchoscopy for removal of these secretions. Chest physical therapy and nasotracheal or endotracheal suctioning were adequate in the other 4. Pneumonia developed in 4 patients with severe obstructive disease. Pneumothorax occurred in 3 other patients, all of whom had bullous disease noted on the preoperative chest roentgenograms.

The nonpulmonary complications included major cardiogenic pulmonary edema in 36 patients, complicated arrhythmias in 32, renal failure in 3, cardiac tamponade in 2, and nonpulmonary infection in 1 patient.

There were two in-hospital deaths, which were attributed to refractory heart failure (patients 1 and 2). One died of progressive heart failure in the ICU 3 days after aortic and mitral valve replacement and the other, of renal and heart failure after a lengthy ICU stay after mitral and tricuspid valve replacement. This amounts to a mortality rate of 12.5% for both subgroups of patients having aortic valve replacement plus another procedure or mitral valve replacement plus another procedure. Neither of these deaths could be directly related to pulmonary complications.

Fig 1. Percent frequency distribution of days spent in the intensive care unit in relation to pulmonary function and surgical procedure. A percent distribution was used rather than a true frequency distribution to account for the differences in population size.



As a group, the average stay in the ICU for patients with severe pulmonary disease was significantly longer (7.9 ± 10.3 days [mean ± standard deviation]) compared with that of the control group of patients without severe pulmonary disease (2.4 ± 3.9 days) ($p < 0.001$). The greater average ICU stay in the study group resulted in part from the fact that 5 patients were in the ICU from 13 to 57 days (Fig 1). Excluding these patients, however, the ICU stay of this group (4.9 ± 2.5 days) was still significantly longer than that of the control group ($p < 0.001$).

To assess whether or not pulmonary complications were responsible for the increase in ICU stay, we arbitrarily divided our population into two groups, those whose ICU stay was 7 days or longer and those whose stay was less than 7 days. Table 2 indicates that patients in the group with the longer ICU stay had nearly twice the number of pulmonary complications as the patients in the shorter-stay group ($p < 0.05$), whereas the incidence of nonpulmonary complications was the same. In addition, Table 2 also indicates that the longer-stay group had significantly worse FEV_1 and maximal voluntary ventilation than the shorter-stay group ($p < 0.005$).

All 5 patients with the longest ICU stays had single- or double-valve replacement procedures. The difference in ICU stay between the patients undergoing bypass grafting and the patients having valve replacement did not reach significance in either the severely impaired group or the control group. To the extent that ICU stay reflects postsurgical complications, however, a trend indicating greater complications after valve replacement was observed in both groups. When the groups were pooled, the ICU stay of the valve replacement patients was more than twice that of the bypass patients (see Fig 1). Furthermore,

Table 2. Comparison of Incidence of Pulmonary and Nonpulmonary Complications and Pulmonary Function Variables by Length of Stay in Intensive Care Unit[a,b]

Length of Stay (d)	FEV_1	MVV	Pulmonary Complications	Nonpulmonary Complications
<7	54.0 ± 9.4	42.9 ± 8.7	1.0 ± 0.9	1.9 ± 0.3
>7	43.3 ± 12.4	32.4 ± 10.2	1.8 ± 1.5	1.9 ± 0.7
p Value	<0.005	<0.005	<0.043	NS

[a] Pulmonary function variables were percent of predicted. [b] Data are shown as the mean ± the standard deviation.

FEV_1 = 1-second forced expiratory volume; MVV = maximal voluntary ventilation; NS = not significant.

Table 3. Results of Simple Correlation Analysis Between Length of Stay in Intensive Care Unit and Pulmonary Function Variables

Variable	r	p	r^2
FVC	−0.420	<0.008	0.176
FEV_1	−0.392	<0.014	0.156
MVV	−0.362	<0.028	0.131
PaO_2	−0.171	NS	0.029
$PaCO_2$	−0.171	NS	0.029
$FEF_{25-75\%}$	−0.060	NS	0.003

$FEF_{25-75\%}$ = mean forced expiratory flow during middle half of FVC; FEV_1 = 1-second forced expiratory volume; FVC = forced vital capacity; MVV = maximal voluntary ventilation; $PaCO_2$ = arterial carbon dioxide tension; PaO_2 = arterial oxygen tension; NS = not significant; r = Pearson correlation coefficient; r^2 = squared multiple.

a χ^2 analysis of the group with severe impairment indicated that the patients who underwent valve replacement had more complications postoperatively than the patients undergoing other cardiac surgical procedures ($p < 0.05$).

To assess the accuracy of pulmonary function tests in predicting postoperative course, a correlation analysis was performed. Table 3 summarizes the results of the simple correlation analysis between length of ICU stay and various pulmonary function variables. Although FVC, FEV_1, and maximal voluntary ventilation each correlated significantly with ICU stay, they accounted for only about 15% of the variance in length of ICU stay (as indicated by the squared Pearson coefficient).

Comment

Over the past few decades, cardiac surgery has become a very common modality in the treatment of various cardiac disorders. Improvements in surgical technique, shortening of intraoperative pump time, and advances in postoperative management including pharmacology, ventilator support, and chest physical therapy [7] have all contributed to a decrease in postoperative pulmonary complications and mortality. Nonetheless, patients with respiratory dysfunction are at increased risk for intraoperative or postoperative pulmonary complications [3, 4]. As cardiac surgical procedures become more commonplace in patients with severe chronic lung disease, the clinician is often faced with the difficult decision of whether to withhold a potentially lifesaving operation because of a prohibitive risk of pulmonary complications.

The value of quantifying the degree of pulmonary function abnormality to assess operative risk is not known in thoracic surgery except before lung resection [8]. Gass and Olsen [5] reviewed the literature concerning selection of specific limits in pulmonary function testing for assessing risks in regard to noncardiovascular thoracic operations. There is little information, however, that considers specific criteria for predicting risk for pulmonary complication after cardiovascular thoracic operations.

We retrospectively evaluated routine pulmonary function test data obtained before cardiovascular operations. From these test results, we identified 39 patients who met our criteria for severe pulmonary dysfunction. The data indicate that, in general, patients undergoing a cardiac procedure do relatively well in spite of tests indicating severely impaired pulmonary function. In the study group, there were no deaths in patients undergoing either bypass grafting alone or single valve replacement. The two deaths occurred in patients having multiple procedures. One patient who died had aortic and mitral valve replacement and the other, mitral and tricuspid valve replacement.

The mortality data from our study population were compared with those of the general population. The mortality rate in the group having bypass grafting alone was comparable with the overall mortality rate of 1% to 2% for patients having bypass grafting at this hospital (hospital statistics). The mortality rate of patients undergoing multiple valve procedures was 12.5% for both the group having aortic valve replacement plus any other procedure and the group having mitral valve replacement plus any other procedure. This compares with an overall rate of approximately 7.0% in a comparably aged group undergoing aortic valve replacement plus another procedure (A. Culliford; personal communication, 1989). We were unable to find published mortality rates for combined mitral and tricuspid valve replacement. For the sake of comparison, however, Gersh and colleagues [9] reported 1 death among 14 patients undergoing combined aortic, mitral, and tricuspid valve replacement, and Czer and associates [10] had a mortality rate of 9.9% for combined mitral valve replacement and bypass. Although the small sample size per procedure precluded our reaching any significantly negative conclusion (ie, there is no difference in mortality rate between the study group and the overall population undergoing a cardiac operation) with confidence, these data suggest that the mortality rate in the study group is comparable with the overall mortality rate.

In addition to the surgical procedure, the nature of the pulmonary disease appeared to contribute to difficulties in the postsurgical course. Patients classified as having restrictive pulmonary disease had fewer respiratory complications than did patients with obstructive disease. This was expected, as chronic airflow obstruction is associated with hypersecretion, secretion retention, increased closing volume, and greater susceptibility to respiratory infections, all of which increase the risk of postoperative complications [11].

In agreement with previous reports [12], these data also indicate that there were more complications after valve replacement than after bypass grafting. Among the patients with severely impaired lung function, there was a disproportionately greater number who underwent valve replacement than bypass grafting compared with the incidence of valve replacement in patients with better pulmonary function. This distribution may reflect the fact that patients with coronary insufficiency and severely impaired lung function might be selected for other types of nonsurgical intervention (eg, peripheral transcutaneous angioplasty), whereas patients requiring valve replacement have fewer alternatives.

It may also reflect the effects of mitral or aortic valve disease, which can cause marked impairment of pulmonary function [13, 14] by increasing pulmonary venous and capillary pressures, which, in turn, can cause pulmonary congestion and interstitial or even frank alveolar edema [15]. Our data suggest that impaired lung function due to the combined effects of valvar disease and primary obstructive pulmonary disease is associated with a higher mortality rate and a more complicated postoperative course than equally impaired lung function entirely attributed to primary obstructive pulmonary disease.

Although on the average, longer ICU stay was associated with poorer preoperative pulmonary function test results, the correlation between FVC and the factors that covary with it (ie, FEV_1 and maximal voluntary ventilation) and postoperative complications, as reflected in length of ICU stay, was weak. Even though these three factors were significant, they account for only a small amount of the variance in observed ICU stays. Thus they fail to clearly define limits below which the hazards of respiratory failure preclude potentially lifesaving cardiac operations.

Pulmonary function tests can serve three purposes. First, in contrast to the opinion of Cain and co-authors [4] that only a good patient history and examination are needed, pulmonary function tests can help identify patients with more severe lung disease than was otherwise suspected on the basis of the personal history and examination. (The importance of a good history and physical examination preoperatively, however, should not be minimized.)

Second, because those patients with severely impaired pulmonary function had, on average, a longer ICU stay and presumably a greater number of pulmonary complications than patients with less severe pulmonary disease [4], pulmonary function tests can help identify patients who are likely to have a more difficult postoperative course so that appropriate preoperative and postoperative management can be planned.

Third, even in patients with less severely compromised pulmonary function, pulmonary function studies can be helpful in detecting airway disease before operation that might be partially reversible with appropriate treatment including bronchodilators and chest physical therapy. For example, there were 10 patients in whom bronchospasm necessitating bronchodilator therapy developed postoperatively, but only 5 had a preoperative history of bronchospasm.

Although these data were collected on a relatively small number of patients, they suggest four important findings. First, although patients with severely impaired pulmonary function do relatively well after a cardiac operation, they are subject to a significantly greater number of postoperative complications than those with less impaired lung function. Second, patients with restrictive lung disease appear to fare better than those with obstructive disease. Third, pulmonary function tests can be used to alert the clinician to possible postoperative complications, but they cannot, by themselves, be used to exclude patients from operation. Fourth, patients with severe pulmonary impairment who are facing valve replacement are at greater risk for death and pulmonary complications than those facing other types of cardiac surgical procedures.

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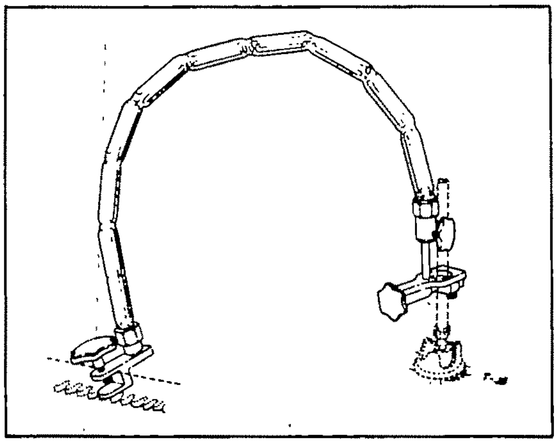

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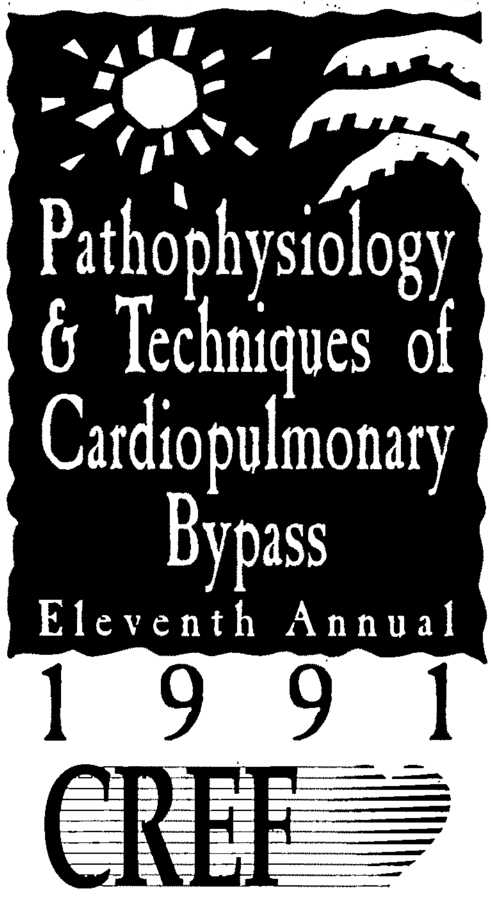

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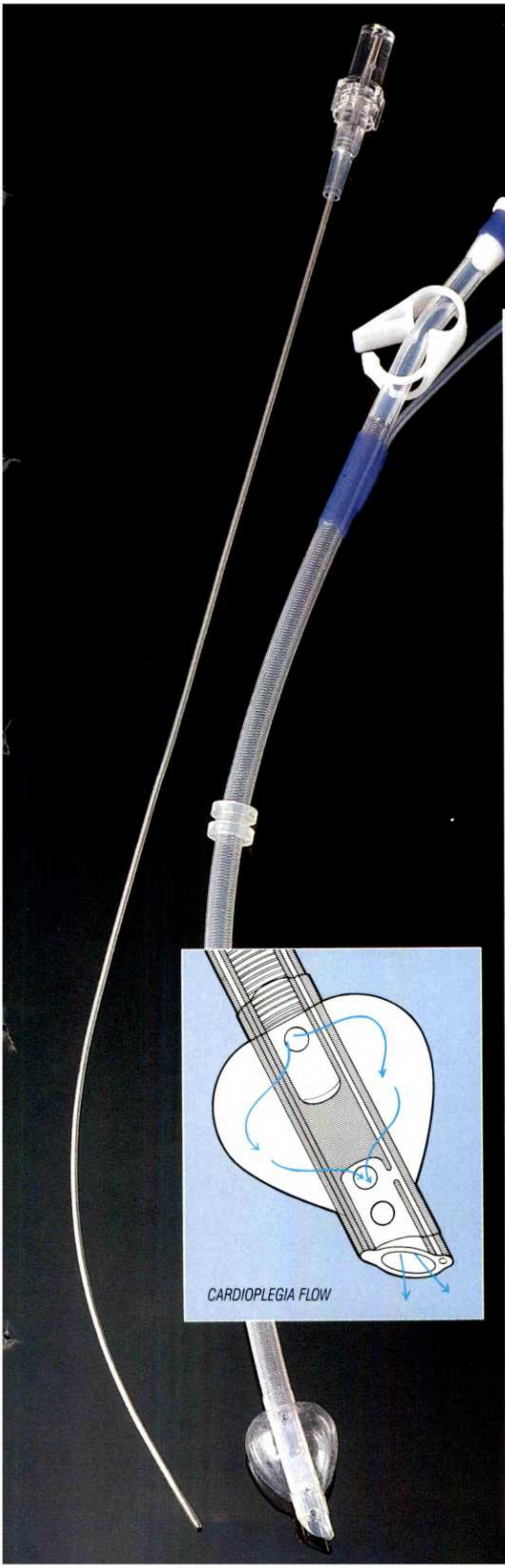

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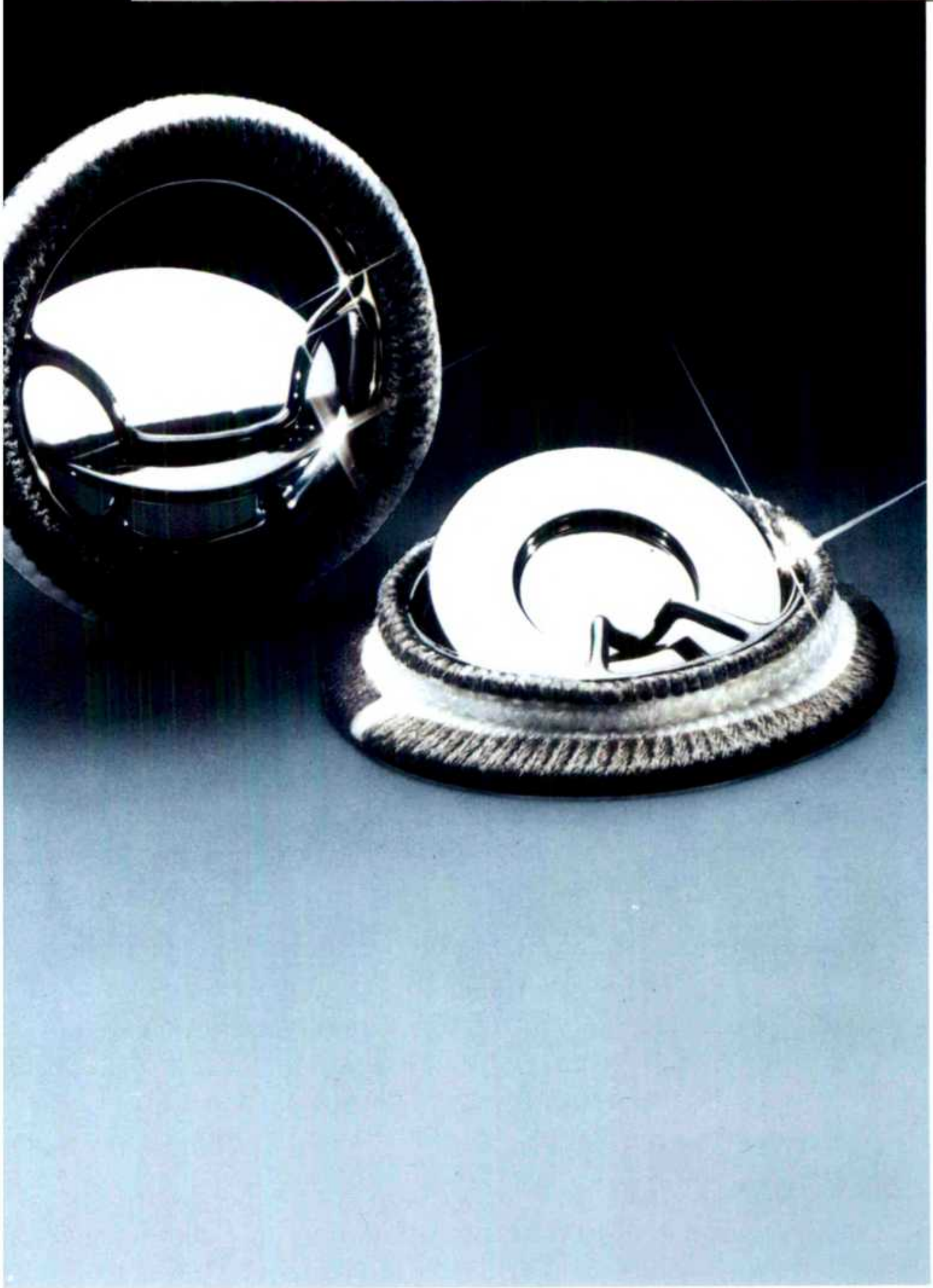

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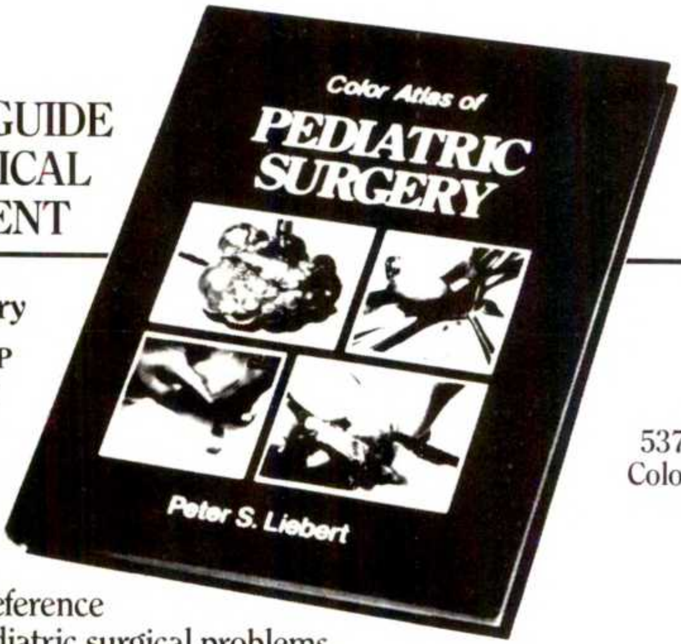

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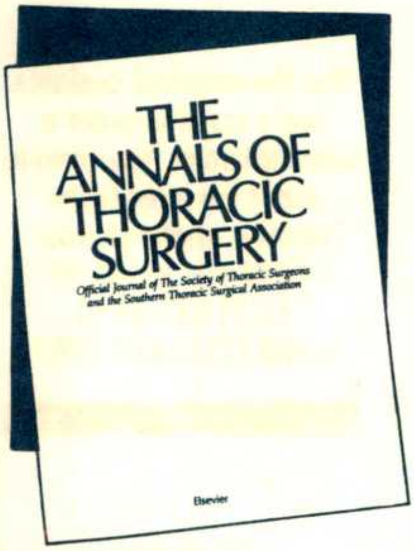
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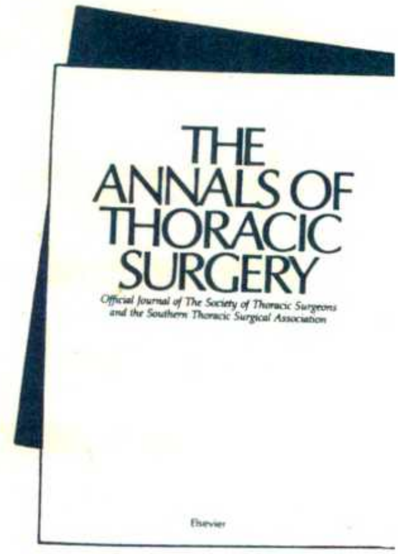

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Results in 104 Patients Undergoing Bronchoplastic Procedures for Bronchial Lesions

Yoh Watanabe, MD, Junzo Shimizu, MD, Makoto Oda, MD, Yoshinobu Hayashi, MD, Sinichiro Watanabe, MD, Ushio Yazaki, MD, and Takashi Iwa, MD

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Bronchoplastic procedures were used in 104 patients with various bronchial disorders. Ten had benign lesions and 94, malignant tumors. The principal operative procedures were sleeve lobectomy and sleeve pneumonectomy for bronchogenic carcinoma, but 11 limited bronchial resections were performed in patients with benign lesions, minute bronchogenic carcinomas, and low-grade malignant tumors. Of the 94 patients with malignant tumors, 79 underwent a bronchoplastic procedure without carinal resection (sleeve lobectomy in 75 and limited bronchial resection in 4), and there was one operative death (1.3%). The overall 5-year survival rate for the patients with bronchogenic carcinoma in this group was 45% and that for patients undergoing curative resection, 57% (survival of patients in stages I, II, and IIIA was 79%, 55%, and 30%, respectively). A bronchoplastic procedure with carinal resection was performed in 15 patients. Twelve in this group underwent sleeve pneumonectomy. There were two operative deaths, and 1 patient has survived for longer than 4 years. Two patients with low-grade malignant tumors underwent carinal resection without lung resection and are still alive. We believe that bronchoplasty is a safe and valuable procedure and that limited bronchial resection appears to be the procedure of choice for localized bronchial lesions.

(Ann Thorac Surg 1990;50:607–14)

Bronchoplasty involves resection of the diseased bronchus followed by reconstruction of the airway and is a parenchymal-sparing operation that was devised in the 1950s as an alternative to pneumonectomy for various bronchial lesions [1]. In the 1960s, sleeve lobectomy was first applied to malignant lung disease [2–4]. Since then, there have been an increasing number of reports on the merits of this method of resection [5–12]. However, some thoracic surgeons are still reluctant to perform bronchoplastic procedures because of technical difficulties and an anticipated increase in complications.

In 1975, we performed our first sleeve lobectomy on a patient with bronchogenic carcinoma. Since that time, we have used bronchoplastic procedures for various bronchial lesions in a series of 104 patients. Most of the patients had bronchogenic carcinoma, and standard sleeve lobectomy was performed as an alternative to pneumonectomy. For benign lesions, low-grade malignant tumors, and minute hilar lung cancers, however, limited bronchial resection has been the procedure of choice at our institution to allow maximal preservation of functional lung tissue. Accordingly, our series includes patients with various bronchial lesions in whom limited bronchial resection was performed with or without pulmonary resection. We have not encountered any increased incidence of complications or worsening of the prognosis with the use of nonstandard bronchoplastic procedures.

This report presents the wide range of benign and malignant diseases for which standard and nonstandard bronchoplastic procedures were used, the types of procedures performed, the associated morbidity and mortality, and the survival rates in a series of 104 consecutive patients.

Material and Methods

A total of 104 patients have undergone bronchoplastic procedures in one institution since 1975. Ninety-four were treated for malignant tumors (89, bronchogenic carcinoma, and 5, low-grade malignant tumors) and 10 for benign disease (7, inflammatory stricture, and 3, benign tumors). The 94 patients with malignant tumors represented 12% of the 778 patients undergoing resection of malignant lung tumors during the same period.

There were 89 male and 15 female patients. Eighty-six of the male patients had malignant tumors, and 3 had an endobronchial lipoma, a bronchial leiomyoma, and a nonspecific inflammatory bronchial stricture. The 15 female patients included 5 with tuberculous bronchial stricture, an infant with nonspecific inflammatory bronchial stricture, 1 patient with bronchial neurofibroma, and 8 patients with malignant tumors. The five tuberculous strictures and the five low-grade malignant tumors were all in female patients. The age distribution by type of bronchial disorder is shown in Table 1. Age of the patients ranged from 20 months to 75 years. Most of the broncho-

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Table 1. Age Distribution by Lesion of Patients Undergoing Bronchoplasty[a]

Bronchial Lesion	Age (y)						
	<20	30–39	40–49	50–59	60–69	70–79	Total
Tuberculous stricture	0	1 (1)	1 (1)	2 (2)	1 (1)	0	5 (5)
Nonspecific inflammatory stricture	1 (1)	0	0	1	0	0	2 (1)
Benign bronchial tumor	0	0	3 (1)	0	0	0	3 (1)
Low-grade malignant tumor	1 (1)	0	1 (1)	2 (2)	1 (1)	0	5 (5)
Malignant bronchial tumor	0	0	5	18 (1)	46 (2)	20	89 (3)
Total	2 (2)	1 (1)	10 (3)	23 (5)	48 (4)	20	104 (15)

[a] Data are shown as the total number of patients; numbers in parentheses are female patients.

plastic operations for benign disease were performed in younger patients.

The histological diagnosis of the malignant bronchial tumors is shown in Table 2. There were 78 (83.0%) squamous cell carcinomas, six adenocarcinomas, two adenosquamous carcinomas, two small cell carcinomas, and one large cell carcinoma. In addition, there were five low-grade malignant tumors (two carcinoids, two adenoid cystic carcinomas, and one mucoepidermoid carcinoma).

Preoperative Management and Selection of Operative Procedure

The initial step in identifying a possible candidate for a bronchoplastic procedure was bronchoscopy. Tomography, computed tomography, and magnetic resonance imaging were also helpful in defining the extrabronchial extent of the lesions. For the patients with tuberculous bronchial stricture, operation was attempted after antituberculosis therapy had been given for more than 6 months. This preoperative treatment caused the tuberculous changes in the bronchus to resolve completely, and there were no cases of recurrence of bronchial tuberculosis around the anastomotic site during follow-up. None of the patients with bronchogenic carcinoma received preoperative radiotherapy. When it was found that the pulmonary artery was involved by extrabronchial tumor, pneumonectomy was preferred over sleeve lobectomy if the pulmonary reserve was adequate. Therefore, in the present series, only 3 patients had sleeve lobectomy combined with circumferential resection of the pulmonary artery.

As the potential cure of the procedure guided the selection of the operation for malignant tumors, sleeve lobectomy and sleeve pneumonectomy were the procedures chosen for most such tumors. However, patients with benign lesions, low-grade malignant tumors, and minute bronchogenic carcinomas underwent limited bronchial resection when complete removal was feasible with this technique (Table 3). Operative procedures used in this series are illustrated in Figure 1. Methods A through C are typical sleeve lobectomies and methods H and I, typical sleeve pneumonectomies. Methods D through G, K, and L are nonstandard procedures designated as limited bronchial resections.

Among the 10 patients with benign bronchial lesions, the 5 with tuberculous bronchial stricture (four right-sided and 1 left-sided) underwent sleeve upper lobectomy, as all 5 also had an upper lobe focus of tuberculosis. The

Table 2. Histological Diagnosis and Sex Distribution of Patients with Malignant Bronchial Tumor[a]

Diagnosis	Male	Female	Total
Squamous cell carcinoma	77	1	78 (83.0)
Adenocarcinoma	5	1	6 (6.4)
Adenosquamous carcinoma	2	0	2 (2.1)
Small cell carcinoma	1	1	2 (2.1)
Large cell carcinoma	1	0	1 (1.1)
Carcinoid	0	2	2 (2.1)
Adenoid cystic carcinoma	0	2	2 (2.1)
Mucoepidermoid carcinoma	0	1	1 (1.1)
Total	86	8	94 (100)

[a] Numbers in parentheses are percentages.

Table 3. Types of Bronchoplastic Procedures for Benign and Malignant Bronchial Lesions[a]

Operative Procedure	Benign Lesion	Low-Grade Malignant Tumor	Bronchogenic Carcinoma	Total
Without carinal resection				89 (13)
Sleeve lobectomy	5 (5)	3 (3)	72 (3)	80 (11)
Limited bronchial resection[b]	5 (2)	0	4 (0)	9 (2)
With carinal resection				15 (2)
Carinal resection with lung resection	0	0	13 (0)	13 (0)
Limited bronchial resection[c]	0	2 (2)	0	2 (2)
Total	10 (7)	5 (5)	89 (3)	104 (15)

[a] Data are shown as the total number of patients; numbers in parentheses are female patients. [b] This includes sleeve bronchial resection without lung resection, sleeve segmentectomy, and second carinal resection without lung resection. [c] This is carinal resection without lung resection.

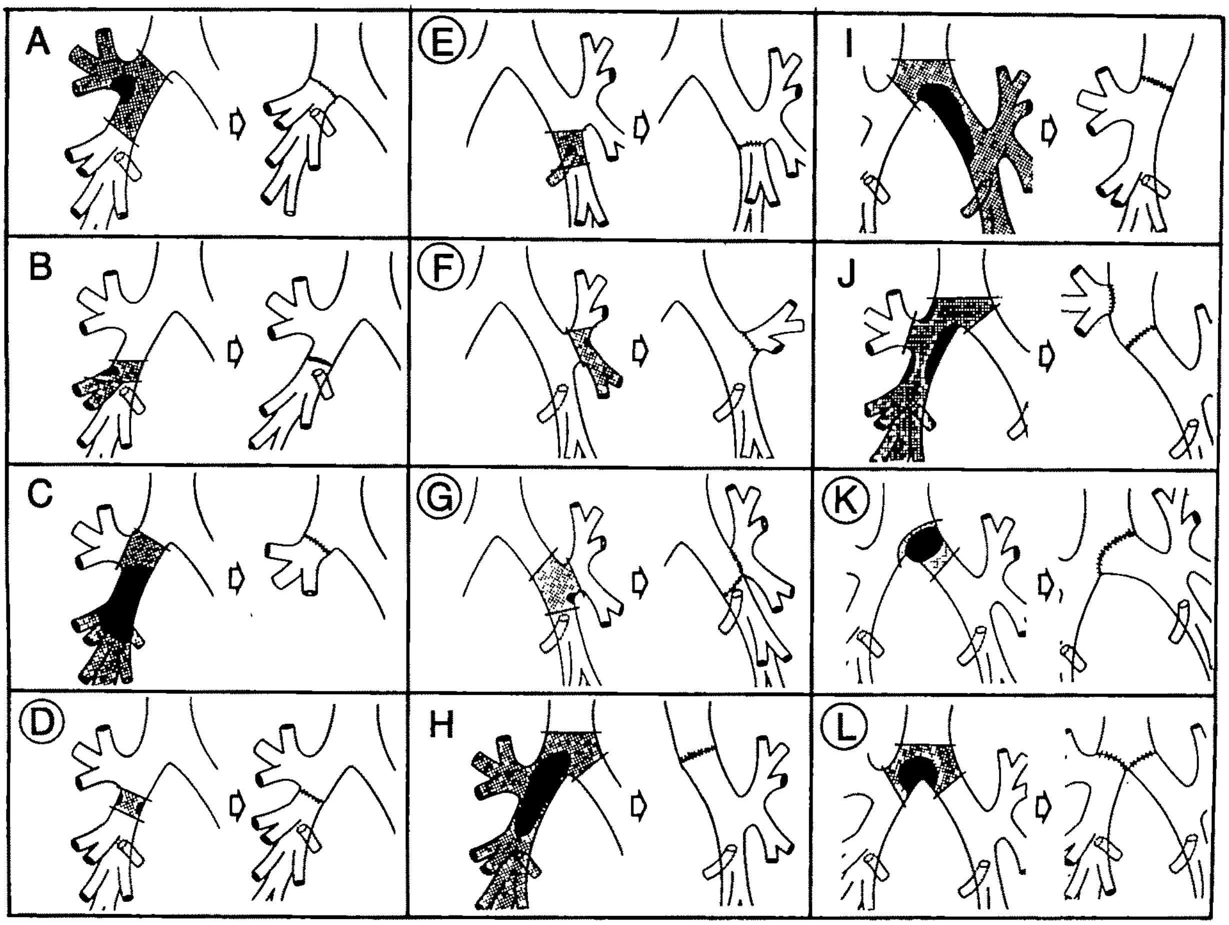

Fig 1. The bronchoplastic procedures used in this series: (A) sleeve upper and lower lobectomy for either lung; (B) sleeve middle lobectomy; (C) sleeve bilobectomy of middle and lower lobes; (D) sleeve resection of intermediate bronchus without lung resection; (E) sleeve segmentectomy of superior segment of lower lobe; (F) sleeve segmentectomy of lingula; (G) second carinal resection without lung resection; (H) right sleeve pneumonectomy; (I) left sleeve pneumonectomy; (J) carinal resection with right middle and lower lobectomy; (K) wedge resection of carina; and (L) carinal resection with double-barreled anastomosis. A circled letter *represents a procedure of limited bronchial resection.*

other five with benign bronchial lesions were treated by limited bronchial resection (see Fig 1, method D). Two patients had stricture of the intermediate bronchus caused by nonspecific inflammatory granulations. The lesion was circumferentially resected and the bronchus joined again by end-to-end anastomosis. One of these patients was a 20-month-old girl with complete obstruction of the intermediate bronchus by granulation tissue. Microscopic examination of the resected specimen confirmed the presence of a foreign body, which might have been vegetable matter. Three patients with benign endobronchial tumors of the intermediate bronchus underwent limited sleeve bronchial resection without lung parenchymal resection.

Bronchoplastic operations for malignant tumors were performed in 94 patients and included 79 bronchoplastic procedures without carinal resection and 15 with carinal resection (see Table 3). The most common procedure in the 79 patients who did not have carinal resection was sleeve lobectomy (75 patients, including 3 who had resection of low-grade malignant tumors: 54 with an involved right lung and 21 with an involved left lung) (Table 4). Limited bronchial resection was applied in 4 patients. Three of them had a minute squamous cell carcinoma at the orifice of a segmental bronchus (one in the superior segment of the right lower lobe, one in the superior segment of the left lower lobe, and one in the lingula of the left lung), and each was completely resectable by sleeve segmentectomy (see Fig 1, methods E and F). In another patient with polypoid squamous cell carcinoma (about 4 mm in diameter) at the bifurcation of the left upper and lower lobe bronchi, second carinal resection without parenchymal resection was performed. The airway was reconstructed by a double-barreled anastomosis between the left main bronchus and the cut ends of the left upper and lower bronchi (see Fig 1, method G).

Fifteen patients with malignant tumors that had invaded the carina or its vicinity underwent carinal resection. Twelve of them had a sleeve pneumonectomy, and 11 of these were right-sided procedures (see Fig 1, method H). In the 1 patient having a left sleeve pneumonectomy (see Fig 1, method I), a median sternotomy was employed instead of a routine posterolateral thoracotomy. The carina and left main bronchus were more accessible through

Table 4. Site of Lesion in 79 Patients With Malignant Bronchial Tumors Undergoing Bronchoplasty Without Carinal Resection

Resected Bronchus	Resected Lung	No. of Patients	Procedure in Figure 1
Sleeve lobectomy			
Right bronchial sleeve resection		54	
Main bronchus	Upper lobe	45	A
Intermediate bronchus	Middle lobe	3[a]	B
Intermediate and main bronchi	Middle and lower lobe	6[b]	C
Left bronchial sleeve resection		21	
Main bronchus	Upper lobe	14[b]	A
	Lower lobe	7	A
Sleeve segmentectomy[c]		3	
Lower lobe bronchus	S6	1	E
Lower lobe bronchus	S6	1	E
Upper lobe bronchus	Lingula	1	F
Second carinal resection[c]		1	
Main, upper, and lower lobe bronchi	None	1	G

[a] One of these patients had adenoid cystic carcinoma. [b] One of these patients had a carcinoid. [c] All of the patients undergoing sleeve segmentectomy or second carinal resection had a minute squamous cell carcinoma without invasion beyond the bronchial wall.

S6 = superior segment of lower lobe.

the median sternotomy approach, which facilitated the operation and probably ultimately minimized operative complications [13]. One other patient with squamous cell carcinoma involving the carina underwent right middle and lower lobectomy with resection of the right main bronchus as well as the carina. An end-to-end anastomosis was performed between the trachea and the left main bronchus plus a side-to-end anastomosis between the trachea and the right upper lobe bronchus (see Fig 1, method J).

In 2 patients with low-grade malignant tumors at the carina, carinal resection without lung resection was done. One patient had mucoepidermoid carcinoma at the orifice of the left main bronchus with invasion into the carina. The thoracic cavity was entered by a right posterolateral thoracotomy, and the left lower part of the trachea, the carina, and part of the left main bronchus were resected so as to remove the tumor without resection of any lung tissue. Side-to-end anastomosis between the trachea and the residual left main bronchus was performed (see Fig 1, method K). The other patient had adenoid cystic carcinoma at the carina. The carina was resected, and reconstruction was performed by a double-barreled anastomosis between the trachea and both main bronchi (see Fig 1, method L).

Operative Technique and Perioperative Management

For ventilatory support during the bronchoplastic procedure, various methods were employed. Balloon catheter occlusion, double-lumen tube intubation, or long tube intubation were used in patients undergoing sleeve lobectomy or sleeve segmentectomy. For all of the patients undergoing carinal resection and some of those undergoing sleeve lobectomy, high-frequency jet ventilation was employed, as previously reported [14]. In some of them, simultaneous double high-frequency jet ventilation was applied using two HFO jet ventilators (Mera Co, Tokyo, Japan).

The suture material used early in the series was nonabsorbable 3-0 Tevdek or Prolene, but in the last three quarters, 3-0 Vicryl was used. The anastomosis was begun at the point farthest from the surgeon. Cartilage-to-cartilage apposition with the placement of two sutures formed the basis of the anastomosis, and further sutures were then added to the cartilaginous part of the bronchus. The membranous part of the bronchus was anastomosed as the last step in the procedure. Luminal disparity was equalized by stretching the membranous portion of the smaller bronchus and crimping that of the larger bronchus. The anastomotic line was routinely covered by a circumferential pleural flap to prevent suture leak and to maintain separation from the pulmonary artery. In a standard sleeve lobectomy or sleeve pneumonectomy for bronchogenic carcinoma, lymph node dissection of the hilar and mediastinal nodes was also performed as in routine lobectomy or pneumonectomy for malignant lung tumors.

As soon as the anastomosis was completed, intraoperative bronchoscopy was performed through an adapter while ventilation was maintained. The anastomotic site was observed, and blood clots and mucus were aspirated from the distal bronchus. Pernasal bronchoscopy was performed at the bedside on the first postoperative day to remove any secretions at the anastomosis and to infuse antibiotics into the reconstructed lung. This procedure was repeated for several days if necessary.

Data Analysis

For the purpose of analysis, the patients were divided into three groups: 10 patients with benign lesions, 79 with malignant tumors undergoing bronchoplasty without carinal resection, and 15 with malignant tumors undergoing carinal resection. Eleven patients (5 with benign lesions and 6 with malignant tumors) underwent limited bronchial resection. Staging was performed by the new international TNM staging system proposed in 1986 [15]. Operations were classified as curative if the tumor and metastatic lymph nodes were completely removed and as noncurative if the tumor, metastatic lymph nodes, or both were incompletely resected. Both operative deaths and noncancer-related deaths during follow-up were included in the calculation of the survival rate. The survival rate was determined by the Kaplan-Meier method, and the Cox-Mantel method was used for the statistical evaluation of differences in survival rate, a p value of 0.05 being considered significant.

Results

Bronchoplasty for Benign Bronchial Lesions

All 5 patients undergoing sleeve lobectomy for bronchial stricture due to endobronchial tuberculosis had a successful operation without complications. They received postoperative antituberculosis therapy for 6 months and had uneventful postoperative courses. These patients are alive and well 4 to 11 years after operation with no evidence of recurrence. Two patients with bronchial stricture resulting from nonspecific inflammatory granulations and 3 with benign endobronchial tumor underwent limited sleeve bronchial resection without lung parenchymal resection. They all had an uneventful postoperative course and are alive and well without any sign of recurrence of bronchial stricture.

Bronchoplasty for Malignant Tumors Without Carinal Involvement

The postoperative staging of the patients with malignant bronchial tumors is shown in Table 5. The 79 patients having operation without carinal resection included 3 with low-grade malignant tumors and 76 with bronchogenic carcinoma. Two of the 3 patients with low-grade malignant tumors were in stage I, and the other was in stage II. Among the 76 patients with bronchogenic carcinoma, 34 had stage I disease, 12 had stage II disease, 28 had stage IIIA disease, and 2 had stage IV disease (both had scattered intrapulmonary metastatic foci in a different lobe on the ipsilateral side, all of which were detected during operation).

There was one operative death (1.3% operative mortality). The patient was a 69-year-old man (stage II) with severely impaired pulmonary function, and death was due to acute respiratory failure on the second postoperative day after sleeve lobectomy.

There were no instances of dehiscence of the anastomosis in this group of patients. However, postoperative complications developed in 2 patients (2.5%). Stricture at the anastomosis, possibly caused by kinking of the bronchus, occurred in 1 (stage I). Another operation was performed, and reanastomosis was successfully achieved by circumferential resection of the right main bronchus. The other patient (stage I) underwent sleeve left upper lobectomy combined with sleeve resection of the pulmonary artery. Thrombosis of the reconstructed pulmonary artery occurred a few days postoperatively, and a completion pneumonectomy was necessary. Metachronous second primary cancers in the opposite lung developed in 2 patients after 5 years and 6 years. At the second operation, 1 patient underwent a second sleeve lobectomy and the other, a standard lobectomy, and neither experienced any postoperative complications.

The prognosis for patients with low-grade malignant tumors was evaluated separately from that for those with bronchogenic carcinoma. Although the one operative death and the three noncancer-related deaths in the follow-up period (one each of chronic obstructive pulmonary disease, cerebral embolism, and pneumonia) were included in the calculation of the survival rate, the patient who had a subsequent completion pneumonectomy was excluded. All the patients with low-grade malignant tumors survived longer than 5 years. The overall (n = 75) 5-year survival rate of the patients with bronchogenic carcinoma was 45%. The survival rate of patients after a curative procedure (n = 64) was 57%, whereas that after a noncurative procedure (n = 11) was poor, and there were no long-term survivors.

Figure 2 shows the survival curves of the patients in each stage who underwent a curative procedure. The 5-year survival rate of stage I patients (n = 33) was 79% and that of stage II patients (n = 12), 55%. For stage IIIA patients (n = 19), the 5-year survival rate was 30% overall, but it was only 17% for patients with N2 disease (a single 5-year survivor to date). Significant differences were noted between stage I and stage IIIA ($p < 0.001$) and between stage II and stage IIIA ($p < 0.05$).

There were 17 5-year survivors in this group of patients. Twelve had squamous cell carcinoma, 2 had carcinoid, and 1 each had adenocarcinoma, small cell carcinoma,

Table 5. Postoperative Staging of Malignant Bronchial Tumors

Stage	Low-Grade Malignant Tumor	Bronchogenic Carcinoma			Overall
		Total	Curative Operation	Noncurative Operation	
Bronchoplastic Procedure Without Carinal Resection					
Stage I	2	34	34	0	36
T1 N0 M0	0	8	8	0	8
T2 N0 M0	2	26	26	0	28
Stage II	1	12	12	0	13
T1 N1 M0	0	3	3	0	3
T2 N1 M0	1	9	9	0	10
Stage IIIA	0	28	19	9	28
T3 N0 M0	0	3	3	0	3
T3 N1 M0	0	5	4	1	5
T1 N2 M0	0	2	1	1	2
T2 N2 M0	0	5	4	1	5
T3 N2 M0	0	13	7	6	13
Stage IV	0	2	0	2	2
T3 N0 M1	0	1	0	1	1
T3 N2 M1	0	1	0	1	1
Total	3	76	65	11	79
Bronchoplastic Procedure With Carinal Resection					
Stage IIIA	0	1	1	0	1
T3 N2 M0	0	1	1	0	1
Stage IIIB	2	12	5	7	14
T4 N0 M0	2	2	1	1	4
T4 N1 M0	0	2	1	1	2
T4 N2 M0	0	7	3	4	7
T4 N3 M0	0	1	0	1	1
Total	2	13	6	7	15

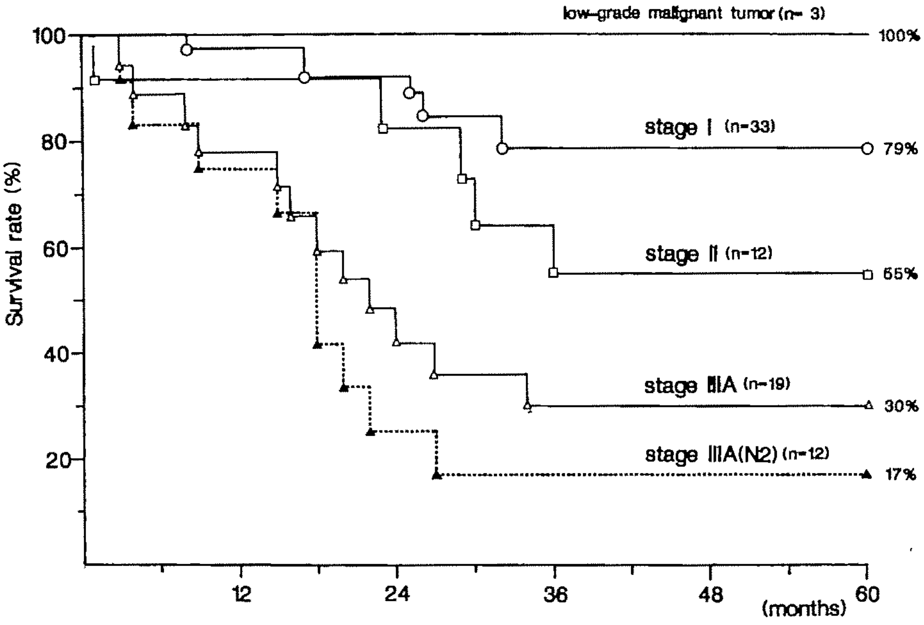

Fig 2. Survival of patients with bronchogenic carcinoma by stage after curative bronchoplastic procedure without carinal resection. Significant differences were noted between stages I and IIIA ($p < 0.001$) and between stages II and IIIA ($p < 0.05$).

and adenoid cystic carcinoma. Eleven had stage I disease; 3, stage II disease; and 3, stage IIIA disease.

Bronchoplasty for Bronchogenic Carcinoma With Carinal Involvement

Carinal resection was performed in 15 patients. Twelve underwent sleeve pneumonectomy, 2 had carinal resection without lung resection, and 1 had carinal resection with right middle and lower lobectomy. Eleven had squamous cell carcinoma, and 1 each had adenocarcinoma, small cell carcinoma (verified by postoperative pathological examination), mucoepidermoid carcinoma, and adenoid cystic carcinoma. The postoperative staging of these patients is shown in Table 5. There were 14 stage IIIB patients and 1 stage IIIA patient.

Figure 3 shows the surgical outcome for these 15 patients. There were two operative deaths after sleeve pneumonectomy. One death was caused by dehiscence of the anastomosis, which occurred on the 25th postoperative day. The other death was caused by a cerebrovascular

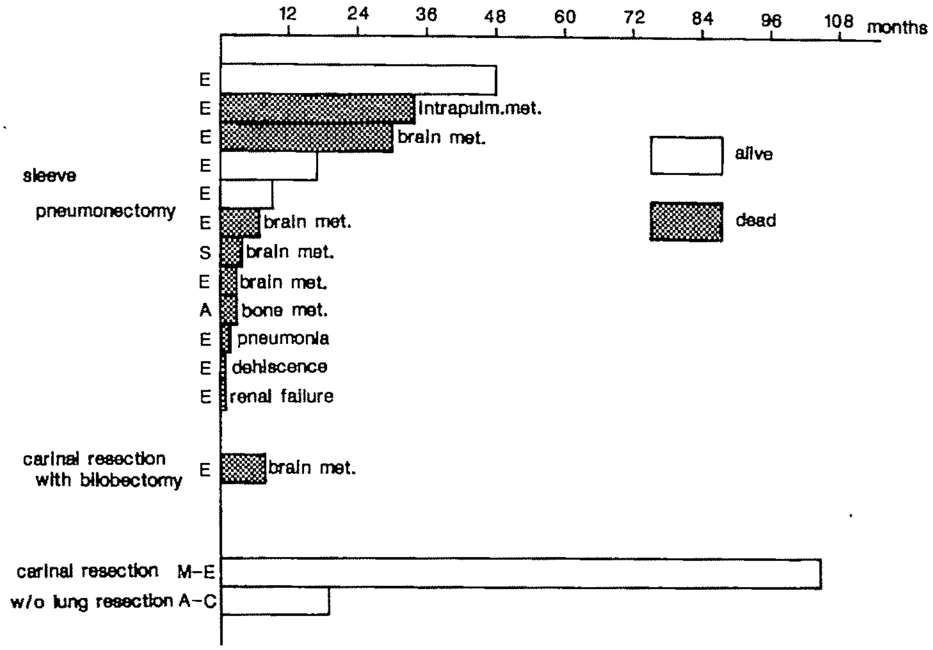

Fig 3. Surgical outcome of patients with bronchogenic carcinoma who underwent a bronchoplastic procedure with carinal resection. Carinal resection without lung resection in patients with mucoepidermoid carcinoma and adenoid cystic carcinoma was done by procedures K and L, respectively, in Figure 1. Carinal resection with bilobectomy was performed by procedure J in Figure 1. (A = adenocarcinoma; A-C = adenoid cystic carcinoma; E = epidermoid carcinoma; M-E = mucoepidermoid carcinoma; met. = metastasis; S = small cell carcinoma.)

accident followed by renal failure after sleeve pneumonectomy combined with replacement of the superior vena cava. There was another case of dehiscence of the anastomosis after sleeve pneumonectomy, which was detected shortly after operation. A minor laceration of the membranous part of the bronchus was successfully repaired by reoperation on the same day.

There were 8 late deaths between 2 and 34 months after operation. Seven patients died of distant metastases (mostly to the brain), and 1 died of aspiration pneumonia. Five patients are still alive; 3 had sleeve pneumonectomy for squamous cell carcinoma, and 2 had low-grade malignant tumors (carinal resection without lung resection). For those having sleeve pneumonectomy, the longest survival is more than 4 years postoperatively. One patient with mucoepidermoid carcinoma (carinal resection without lung resection) is alive and well 8 years 8 months after operation without any sign of recurrence. Another patient had carinal resection without lung resection for adenoid cystic carcinoma at the carina. Although extensive resection of the right main bronchus was performed as far as the orifice of the right upper lobe bronchus, microscopic residual cancer at the resected margin of the right main bronchus was detected by postoperative examination of the resected specimen. Postoperative radiotherapy was given, and this patient is alive and disease free 19 months postoperatively.

Comment

Sleeve lobectomy is an alternative to pneumonectomy and provides an improved quality of life to the patient. In addition, this procedure, which maximizes residual lung tissue, can allow a second procedure in patients with second primary cancers of the lung, the incidence of which has increased in recent years [16]. Among the 93 patients having bronchoplasty for bronchogenic carcinoma in our series, 2 patients had a second operation for metachronous primary cancer that developed more than 5 years postoperatively.

Furthermore, limited bronchial resection with or without pulmonary parenchymal resection allows maximal lung conservation in patients with benign bronchial lesions and in select patients with malignant tumor (low-grade malignant tumors and minute early hilar carcinoma without invasion of extrabronchial tissue). We preferentially performed limited bronchial resection whenever feasible, and it was used in 11 of the 104 patients in this series. These 11 patients included 2 with nonspecific inflammatory bronchial stricture, 3 with benign endobronchial tumor, 4 with minute hilar squamous cell carcinoma, and 2 with low-grade malignancies at the carina. Neither complications nor recurrences were encountered after nonstandard limited bronchial resection in these patients, and postoperative ventilation/perfusion scans showed complete preservation of pulmonary function. When limited bronchial resection for bronchogenic carcinoma is performed, endoscopic observation and biopsy are crucial in delineating the extent of the disease and planning the site of resection, a decision that cannot be made as well by observing the outside of the involved bronchus at operation. We emphasize that when this procedure is used for bronchogenic carcinoma, it should be restricted to minute squamous cell carcinoma without extrabronchial growth or nodal involvement.

Toomes and Vogt-Moykopf [11] reported that 88 patients underwent combined bronchoplastic and angioplastic procedures with an operative mortality of 17%. We intentionally selected pneumonectomy rather than such combined procedures if pulmonary function was adequate. The operative mortality among the patients undergoing pneumonectomy is 5.8% (7/121 patients) in the 15 years since the first bronchoplastic procedure was performed at our institution. To date, only 3 patients who have undergone combined bronchoplastic and angioplastic operations. One of these patients had squamous cell carcinoma and underwent sleeve left upper lobectomy with angioplasty of the left main pulmonary artery. Six years later, a second primary cancer developed in the opposite upper lobe. At the second operation, he underwent right upper lobectomy without any postoperative complications. Our experience indicates that if the surgical candidate is properly selected from among those with squamous cell carcinoma, preservation of lung function to allow future procedures can be achieved.

The 5-year survival rate of patients with bronchogenic carcinoma undergoing resection (n = 778) at our institution in the past 15 years is 39% overall and after curative resection (n = 516), 52%. On the other hand, the overall 5-year survival rate and that after a curative procedure for patients having bronchoplasty without carinal resection were 45% and 57%, respectively, a more favorable prognosis than that afforded by the other methods of resection such as standard lobectomy or pneumonectomy. These figures are comparable with the reports of 30% by Faber and colleagues [5], 34% by Bennett and Abbey-Smith [6], 41% by Van Den Bosch and associates [8], 41% by Naruke [7], 45% by Frist and co-workers [9], and 50% by Ungar and colleagues [10]. The results of our study support the contention that bronchoplastic resection of bronchogenic carcinoma does not compromise the adequacy of the resection.

The 5-year survival rate of patients with bronchogenic carcinoma in stages I and II was 79% and 55%, respectively, showing favorable results. The 5-year survival rate of patients in stage IIIA undergoing a curative procedure was 30% overall, but it was only 17% for the group with positive metastatic lymph nodes in the mediastinum (1 patient survived 5 years). Van Den Bosch and associates [8] reported that no patient with positive hilar nodes survived long term among 8 of 22 patients who underwent sleeve lobectomy. They concluded that the finding of positive hilar nodes contraindicated sleeve resection. In such cases, when pneumonectomy is impossible from a functional point of view, sleeve resection should be regarded as a palliative procedure. However, Faber and associates [5] stated that positive lobar or mediastinal nodes identified at the time of operation do not contraindicate a sleeve lobectomy. They reported that the 5-year survival was 23% for patients with positive nodes who did not have radiotherapy and 15% for stage III patients. Based on these data, they believed that sleeve lobectomy could be a satisfactory procedure even when the disease was advanced. Naruke [7] recently reported a 33% 5-year survival rate in N2 patients undergoing bronchoplasty. These data have encouraged us to still consider bronchoplastic procedures for patients with squamous cell carcinoma with limited mediastinal lymph node involvement.

The reported operative mortality after sleeve resection of the bronchus for bronchogenic carcinoma ranges from 0% to 11% [5–12]. In our present series, the operative mortality after sleeve resection without carinal resection was 1.3%, which is acceptable for surgical intervention in patients with lung cancer. Concerning postoperative complications of the reconstructed airway, such as fistula or stricture, a range of incidences has been reported [11]. In this series, the rate of postoperative complications was 2.5% (2/79 patients), which is relatively low compared with the percentages in the preceding reports. In particular, the incidence of postoperative stricture caused by granulations was extremely low. This favorable outcome might have been achieved by careful performance of the anastomosis, the choice of suture material, and the bronchial toilet performed using bronchoscopy in the postoperative period. The survival, mortality, and morbidity rates in this series strongly support the conclusion that bronchoplastic procedures are indicated when technically feasible, even if the patient can tolerate a pneumonectomy. Such procedures are especially applicable to localized lesions that do not involve great loss of lung parenchyma.

As for sleeve pneumonectomy, many problems remain unresolved. As was pointed out by Deslauriers and associates [17], the surgical indications, technique, and even the prospects for cure are all unclear because very few institutions have a large experience with this procedure. That group performed 38 sleeve pneumonectomies for lung cancer over a 20-year period (1967 to 1987) with an almost 50% rate of major postoperative complications and a 29% operative mortality. Jensik and co-workers [18] also reported an operative mortality after sleeve pneumonectomy of 29% (10/34 patients). In our series, there were two operative deaths among the 12 patients having sleeve pneumonectomy (an operative mortality of 17%), one death resulting from dehiscence at the anastomosis and one not related to the surgical technique. The 5-year survival rates reported by Deslauriers and co-workers [17] and Jensik and associates [18] were 13% and 15%, respectively. We began using this operative procedure in 1983 and hence have no 5-year survivors yet, but 1 patient (squamous cell carcinoma without nodal metastasis) has survived for more than 4 years after operation. Most of the deaths were due to distant metastases, and the chief site of metastasis was the brain. Based on these results, we suggest that sleeve pneumonectomy be confined to patients with squamous cell carcinoma with minimal nodal involvement.

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Pharyngoesophageal Perforation After Blunt Neck Trauma

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Pharyngoesophageal perforation secondary to blunt neck trauma is an uncommon injury that can cause serious morbidity and mortality if not recognized and treated. Pharyngeal perforation secondary to blunt trauma sustained while boxing is reported. Review of the world literature found 10 cases of pharyngoesophageal perforation secondary to blunt neck trauma. Analysis of these cases indicates that perforations less than 2 cm and limited to the pharynx may be treated medically with close observation. Large perforations and those perforations that extend to the esophageal inlet or involve the esophagus exclusively are best treated surgically.

(Ann Thorac Surg 1990;50:615–7)

Perforation of the pharynx or esophagus can result from many causes, including blunt external trauma to the chest and neck, penetrating wounds, iatrogenic trauma during operation, endoscopy, or intubation, swallowed foreign bodies, blast injury, and postemetic perforation as seen in Boerhaave's syndrome [1]. Perforation after nonpenetrating, external blunt neck trauma is an uncommon injury, with only 10 cases reported in the literature. Recently, a patient who sustained blunt neck trauma during a boxing match was evaluated at Walter Reed Army Medical Center and found to have a pharyngeal perforation. This unique injury prompted an extensive literature search. Analysis of the compiled cases provides useful guidelines for the management of perforations following blunt neck trauma.

Case Report

The patient is an 18-year-old man who sustained blunt anterior neck trauma during boxing competition at the United States Military Academy. He experienced no immediate posttraumatic symptoms; however, odynophagia and dysphagia developed 48 hours after the event. On presentation for medical care the patient was afebrile. Physical examination showed crepitus of the anterior neck at the level of the hyoid and larynx. Plain neck roentgenograms were remarkable for subcutaneous and retropharyngeal emphysema. On the basis of clinical and radiographic findings a diagnosis of pharyngeal perforation, less than 1 cm in length, was made. The patient was hospitalized and given intravenous antibiotics (Penicillin G, 2 million units every 4 hours), and oral feedings were withheld (*nil per os*). He remained afebrile during his hospital course and tolerated clear fluids on postperforation day 6. The patient was discharged well on day 7 and has had no difficulties during the follow-up period of 1 year.

Comment

Esophageal perforation after external blunt trauma is a rare injury. Worman and Hurley [2] reviewed the English-language literature and found only 30 cases reported between 1900 and 1962. Pharyngoesophageal perforation secondary to blunt trauma to the neck is even more infrequent, accounting for less than 2% of all perforations of this anatomic region [3]. Our case report describes pharyngeal perforation secondary to blunt neck trauma sustained while boxing, bringing the total number of reported cases of pharyngoesophageal perforation after blunt neck trauma to 11.

These 11 cases can be divided into two categories depending on the method of management: either nonoperative therapy or surgical repair. The first category of therapy is nonoperative or conservative medical management [4, 5]. This is recognized by most authors as keeping the patient *nil per os* with nasogastric drainage, administering intravenous fluids and broad-spectrum antibiotics, and, finally, hyperalimentation if necessary. In this series 5 patients were managed in this manner. The second modality of management is primary surgical closure. This usually consists of surgical exploration, debridement if necessary, and suture closure of the perforation [3, 6, 7]. Adequate drainage is an important consideration. Surgical closure was the management option for 6 of the 11 patients, and all 6 had retropharyngoesophageal drains placed before skin closure.

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The opinions expressed are those of the authors and are not intended to represent official policy of the United States Air Force, the United States Army, or the Department of the Defense.

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Table 1. Cases of Pharyngoesophageal Perforation Managed Nonoperatively

Author	Cause of Perforation	Location	Size (cm)	Diagnosis (h)	Outcome
Hagan [8]	Blunt neck trauma (steering wheel)	Pharyngoesophagus	1	<12	Abscess/ discharge
Hagan [8]	Blunt neck trauma (kick to neck)	Pharynx	1	<12	Uneventful recovery
Wind [9]	Blunt neck trauma (fall onto edge of bed)	Pharynx	1	<12	Uneventful recovery
Yates [10]	Blunt neck trauma (steering wheel)	Pharynx	2	<12	Uneventful recovery
Niezgoda et al [present report]	Blunt neck trauma (boxing injury)	Pharynx	1	>12	Uneventful recovery

Analysis of the data reported by each author provides useful guidelines for the management of these injuries. Four of the 5 patients in the nonoperative group enjoyed an uneventful recovery (Table 1). These patients sustained perforations that were small in size, less than 2 cm, and were limited to the pharynx. In the fifth patient in this group, an abscess developed that required surgical drainage and wound care of the abscess cavity for 3 days. The patient was discharged 6 days after drain placement. This patient sustained a perforation not limited to the pharynx but involving the esophagus.

In comparison, 3 patients with perforation limited to the pharynx were treated surgically and did well (Table 2). These 3 patients, in contrast to the patients treated medically, suffered larger perforations, greater than 2 cm. The remaining 3 patients in the surgical group were treated for perforations that either extended into the esophagus or were limited to the cervical esophagus. All 3 patients recovered; however, in 1 patient in whom the diagnosis was delayed for greater than 12 hours a paraesophageal abscess developed, which was managed with cervical esophagostomy and debridement. This patient was discharged well after closure of the esophagostomy.

These data suggest that medical therapy be reserved for perforations that are less than 2 cm and are limited to the pharynx. Surgical repair and drainage should be standard treatment for larger perforations or for those perforations that involve the esophagus. Therapy based on these guidelines does not appear to be influenced by a delayed diagnosis, but as in all cases of pharyngoesophageal perforations, a timely diagnosis favors a successful outcome.

These cases also provide interesting data regarding associated radiographic findings. Ten of the 11 patients had evidence of retropharyngeal or other subcutaneous emphysema on plain cervical roentgenograms. This suggests that abnormal air streaking seen on plain films after blunt neck trauma is nearly diagnostic for perforation and should cause a high degree of clinical suspicion (Fig 1). Any patient who sustains serious blunt neck trauma should receive a lateral cervical spine roentgenogram as part of the evaluation in the emergency room.

Pharyngoesophageal perforation after nonpenetrating, external blunt neck trauma is an uncommon injury. A high index of suspicion in combination with a detailed evaluation of cervical roentgenograms and knowledge of the size and location of the perforation will optimize successful treatment of this otherwise difficult diagnostic and management dilemma.

Table 2. Cases of Pharyngoesophageal Perforation Managed Surgically

Author	Cause of Perforation	Location	Size (cm)	Diagnosis (h)	Outcome
Gulbrandson and Gaspard [11]	Blunt neck trauma (steering wheel)	Pharynx	4	<12	Uneventful recovery
Tucker and Padula [12]	Blunt neck trauma (fell onto forearm)	Pharynx	3	<12	Uneventful recovery
Hagan [8]	Blunt neck trauma (steering wheel)	Pharynx	3	<12	Uneventful recovery
Hagan [8]	Blunt neck trauma (steering wheel)	Pharyngoesophagus	4	<12	Uneventful recovery
Lerschlolam and Banchuin [14]	Blunt neck trauma (elevator blow to occiput)	Cervical esophagus	. . .[a]	<12	Uneventful recovery
	Blunt neck trauma (steering wheel)	Cervical esophagus	. . .[a]	>12	Delayed recovery

[a] Not reported.

control and short-term survival are better in those patients treated with postoperative irradiation.

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Use of the Variable-Length Intraluminal Sutureless Graft

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From August 1987 to May 1988 we treated 4 patients with acute ascending aortic dissections with a variable-length intraluminal aortic prosthesis. This operation uses profound hypothermic circulatory arrest and represents a refinement of existing techniques. There was no mortality, and morbidity was minimal. Modifications of this technique can be used in performing proximal aortic root reconstruction with a composite valved conduit. The use of a variable-length intraluminal prosthesis and hypothermic circulatory arrest is illustrated. This is a safe and useful technique in select cases of acute ascending aortic dissection.

(Ann Thorac Surg 1990;50:624–30)

Operative therapy of acute ascending aortic dissection is associated with a lower mortality than medical therapy [1, 2]. Nevertheless, operative mortality remains high because of hemorrhage from suture lines and myocardial failure related to prolonged cardiopulmonary bypass and massive transfusion. Berger and others [3] proposed repairing acute dissection with the intraluminal sutureless prosthesis in hopes of achieving a more expeditious and hemostatic anastomosis. Despite initially gratifying results there have been serious technical and anatomical difficulties with the commercially available fixed-length devices. The effective use of this vascular substitute requires a thorough understanding of its limitations.

The concept of sutureless fixation by circumferential ligature over a rigid intraluminal graft was first suggested by Carrel [4] in 1912 and later adopted by Hufnagel [5]. Despite concerns about possible erosion, vessel wall necrosis, and prosthesis migration, several investigators independently developed sutureless prosthetic conduits and demonstrated that they could be used in replacement of the thoracic aorta [6–8]. These devices became available commercially for human implantation in 1980. Since the devices initially available were fixed in length, their use frequently required either sacrificing one of the fixation "spools" and hand-sewing one anastomosis or inappropriately adjusting the extent of aortic exclusion.

We report here the use of the variable-length intraluminal graft (Intraaortic woven Dacron graft with Vari Length; Meadox Medicals, Inc, Oakland, NJ) in repair of acute ascending dissection with resuspension of the aortic valve. We have found use of this prosthesis with hypothermic circulatory arrest [9] to be a simple and safe technique for select patients with acute ascending aortic dissection.

Material and Methods

Patients

Between August 1987 and May 1988 4 patients were operated on using the technique illustrated. There were 2 women (aged 71 and 75 years) and 2 men (aged 61 and 63 years). In each patient, symptoms of acute dissection had developed less than 24 hours before admission, and each had some degree of aortic insufficiency. All patients underwent cardiac catheterization and echocardiography before operation. In 1 patient arterial access to the true lumen of the ascending aorta could not be obtained and magnetic resonance imaging was performed instead. The other 3 had preoperative coronary angiography and an aortic root injection. Operation was undertaken directly after the diagnosis was confirmed.

Technique of Repair

The anesthetic management is similar to that employed for elective cardiac operations. Body temperature is monitored by esophageal, pulmonary artery, and urinary bladder thermistors. A pulmonary artery catheter, left radial artery catheter, and urinary catheter are inserted before induction of anesthesia. The ambient room temperature is lowered to between 16.7° and 18.3°C (62° and 65°F). The head is packed in ice immediately after induction of anesthesia and before draping. Shortly before the circulation is arrested thiamylal (30 mg/kg), mannitol (25 g), furosemide (40 mg), and methylprednisolone (2 g) are administered [10].

Cardiopulmonary bypass is established using a 22F femoral arterial cannula and a 51F two-stage atrial cannula. The pulmonary artery is vented with an 18F sump catheter if the aortic insufficiency is mild (Fig 1). If there is moderate or severe aortic insufficiency, a right superior pulmonary vein vent should be used. A cardioplegia cannula is inserted into the coronary sinus (Retroplegia coronary sinus cardioplegia cannula; Research Medical, Inc, Salt Lake City, UT) via a pursestring suture in the

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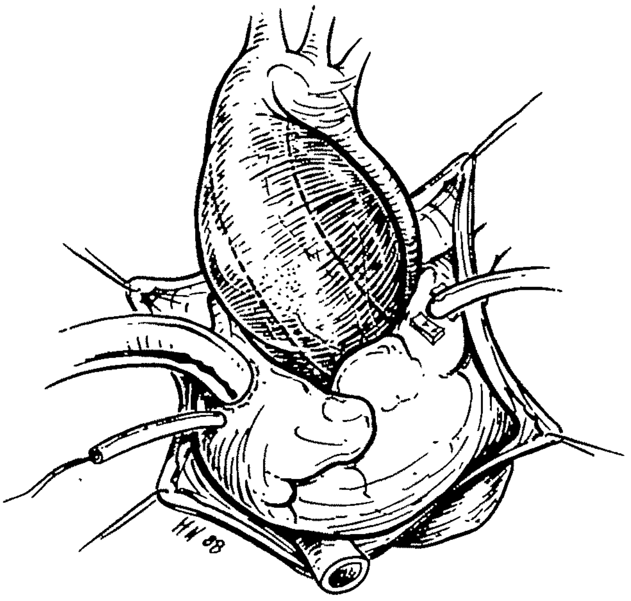

Fig 1. Operative appearance of acute ascending dissection after cannulation for bypass. A right superior pulmonary vein cannula may be substituted for the pulmonary artery sump (illustrated) if there is moderate or severe aortic regurgitation.

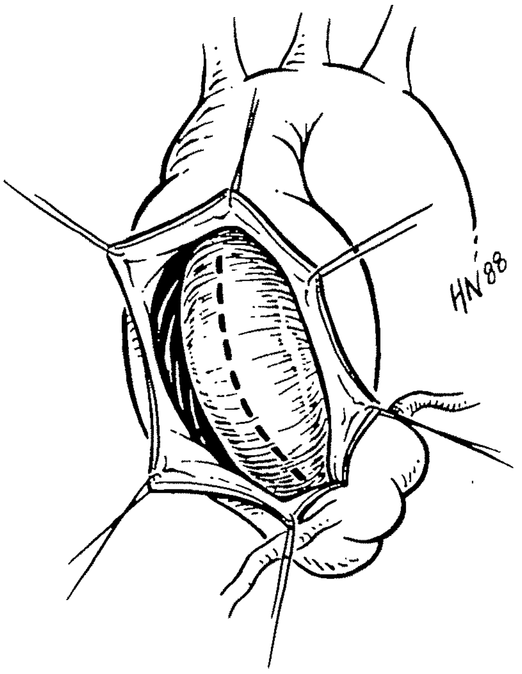

Fig 2. With the circulation arrested the aorta is opened longitudinally. Thorough inspection of the true and false lumens helps plan the repair.

right atrial wall. A septal temperature probe is used to insure adequate myocardial cooling. We have chosen to cool the circulation to 19°C for all patients as this allows a safe period of circulatory arrest during which a comprehensive evaluation of the entire aortic root, ascending aorta, and arch may be performed.

During cooling the aorta is dissected free of the pulmonary artery and pericardium. The aorta is encircled with Dacron umbilical tapes at the innominate artery and 1 cm above the coronary ostia. Intermittent reduction of pump flow can be helpful in exposing the fragile aortic wall posteriorly and medially.

When all temperature probes have reached the target temperature the patient is placed in steep Trendelenburg position and the circulation is arrested. Although air may enter the arch itself the arch vessels will remain filled and need not be individually clamped. A forward flow of 25 mL/min from the femoral artery catheter may help keep the blood level constant in the arch.

With the venous circuit exsanguinated the aorta is opened longitudinally and the site of intimal disruption is sought (Fig 2). The intraluminal technique should be considered if the dissection involves the ascending aorta with sparing of the coronary ostia and with little or no dilation of the aortic arch. Modifications of the technique can be used if a composite valved conduit is used for proximal reconstruction (see Comment section).

It is frequently possible to determine by echocardiography or by external inspection of the aorta if a composite valved-graft reconstruction of the aortic root will be necessary before operation. Patients with Marfan's syndrome and those with long-standing aortic insufficiency or annuloaortic ectasia should undergo valved-conduit replacement because simple resuspension of an anatomically abnormal or dilated aortic valve will not restore competence. The aortic regurgitation accompanying ascending dissection results from loss of suspension of one or more commissures by the enlarging mural hematoma. A composite-valved conduit may also be necessary if there is proximal dissection involving the coronary ostia or the sinus segment of the aorta. If it is determined that a composite graft replacement will be necessary, such as for Marfan's syndrome, then the distal ascending aorta is clamped with an atraumatic clamp and retrograde cardioplegia is delivered during cooling so that the proximal portion of the repair can be performed first.

When resuspension can be performed it is carried out by placing a single Teflon-pledgeted 3-0 polypropylene suture at the apex of each commissure perpendicularly through the aortic wall (Fig 3).

The Meadox intraaortic woven Dacron graft with Vari Length is constructed of low-porosity woven Dacron (50 $\text{mL} \cdot \text{cm}^{-2} \cdot \text{min}^{-1}$). It is supplied in a variety of external diameters. The prosthesis comes supplied with one rigid intraluminal spool fixed to the end and a second movable spool of equal diameter. After the appropriate diameter graft is selected the surgeon must carefully suture the movable ring to the Dacron tube at the desired length. Two Dacron braided ligature tapes are also supplied to allow the external aortic wall to be circumferentially ligated to the rigid spools. The graft may be preclotted with nonheparinized blood, but we have not found this step essential during ascending aortic replacement. Excessive instrument handling of the graft after preclotting will disturb the yarn bundles and interstitial fibrin.

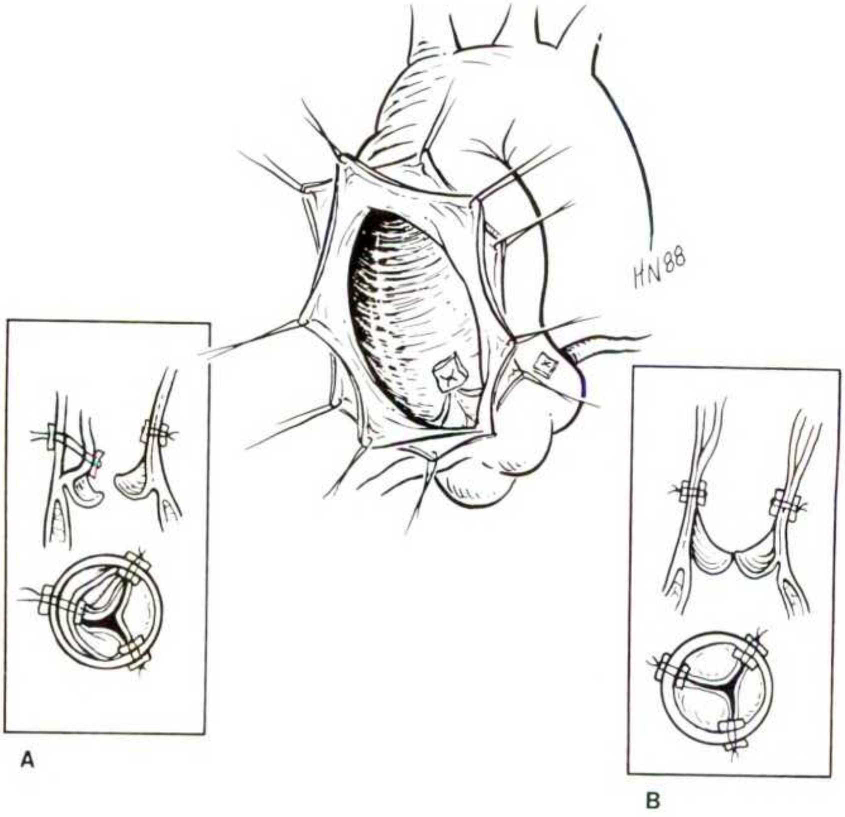

Fig 3. The technique of aortic commissural resuspension is illustrated. Aortic competence will be restored if the aorta is not excessively dilated at the level of the commissures and if the sutures are precisely placed.

Valve sizers are used to calibrate the aortic root and the distal ascending aorta. We have generally selected a prosthesis at least 2 mm smaller than the internal aortic diameter. The distal spool is implanted first. Two fixation sutures of 3-0 polypropylene are passed through the *proximal* fabric of the spool and then through the *excluded* portion of the aortic wall 1 cm proximal to the innominate artery (Fig 4). The Dacron tape is tied firmly but not excessively tightly over the ridge in the spool. The assistant can stabilize the first throw with a small hemostat as the knot is completed. After six throws a small hemoclip is used to prevent the ends of the Dacron from unraveling. If the arrest period is greater than 30 minutes at this point, the graft can be clamped and the pump may be

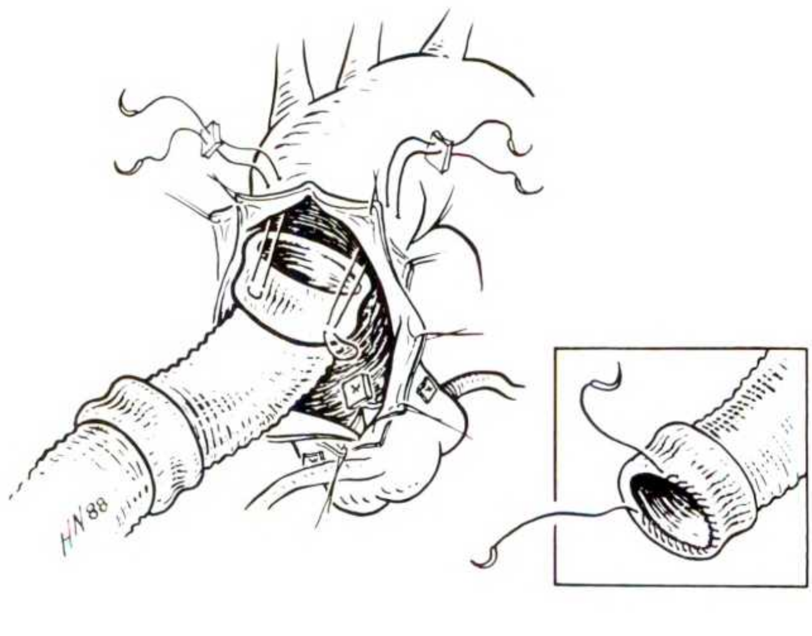

Fig 4. The distal spool of the prosthesis is positioned in the distal ascending aorta with two pledgeted sutures. It is important to place these sutures through only the excluded *portion of the aortic wall to avoid bleeding. (Inset) The proximal (mobile) spool is fixed to the graft with a running suture after the length of the graft is adjusted.*

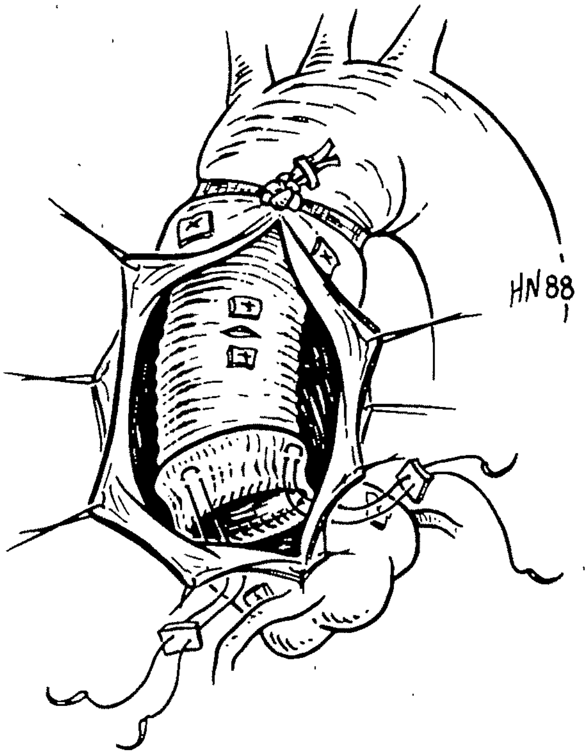

Fig 5. In positioning the proximal spool occlusion of the right coronary ostium must be avoided. The graft may be clamped and cardiopulmonary bypass restarted during proximal spool fixation if the limit of safe circulatory arrest is approaching.

restarted. It is technically easier, when the ascending aorta is short, to fix both the proximal and distal spools with the circulation arrested as the cross-clamp may dislodge the distal spool.

The graft is trimmed to appropriate length with a small hand-held ophthalmic cautery and the mobile spool is fixed to the cut end of the Dacron with a running stitch of 3-0 polypropylene. The proximal spool is positioned and fixed in place using a similar technique (Fig 5). Care must be taken to avoid obstruction of the right coronary ostium. Before flow is restored the graft is vented in its midportion to allow evacuation of air (Fig 6). Systemic warming is commenced as soon as the pump is restarted. During the rewarming interval the aortic wall is closed over the graft with Teflon felt strips or pledgets (Fig 7). At the distal spool a "girdle" is constructed by snugly wrapping the aorta with a strip of Teflon felt (USCI Teflon felt; CR Bard Inc, Billerica, MA) 2 × 15 cm. A vascular stapler (Proximate reloadable linear stapler (30 mm), RLV 30 vascular; Ethicon, Somerville, NJ) is used to secure the band of felt while the aorta is decompressed by reducing pump flow (Fig 8). A small defect in the aortic closure is left until after protamine is administered. As the graft has not been preclotted there is considerable interstitial oozing.

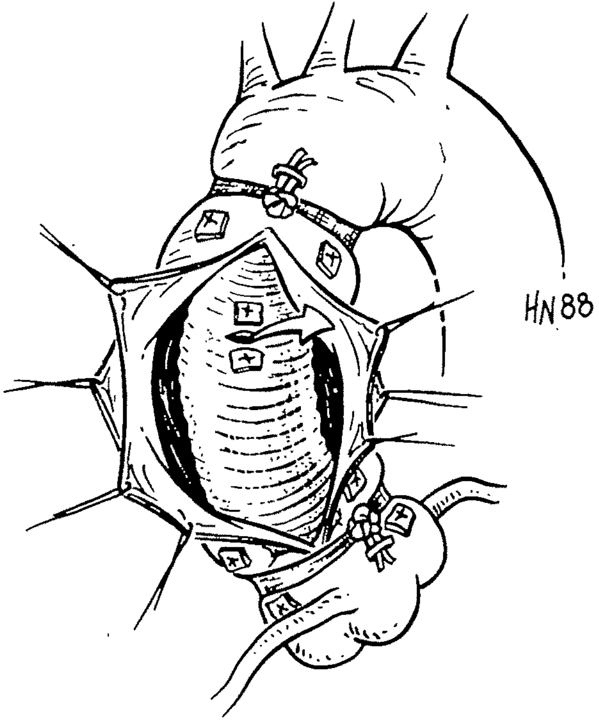

Fig 6. The prosthesis is vented to evacuate air. The aortic commissural sutures are the only transmural *sutures placed during the repair.*

When normothermia is reached the pulmonary sump is removed and cardiopulmonary bypass is slowly discontinued. Five grams of ϵ-aminocaproic acid are administered after bypass is discontinued. After the protamine is given, desmopressin acetate (DDAVP injection; Rorer Pharmaceuticals, Fort Washington, PA) (0.3 μg/kg) is administered. All patients receive platelet transfusions. Fresh frozen plasma and cryoprecipitate are not routinely given unless indicated.

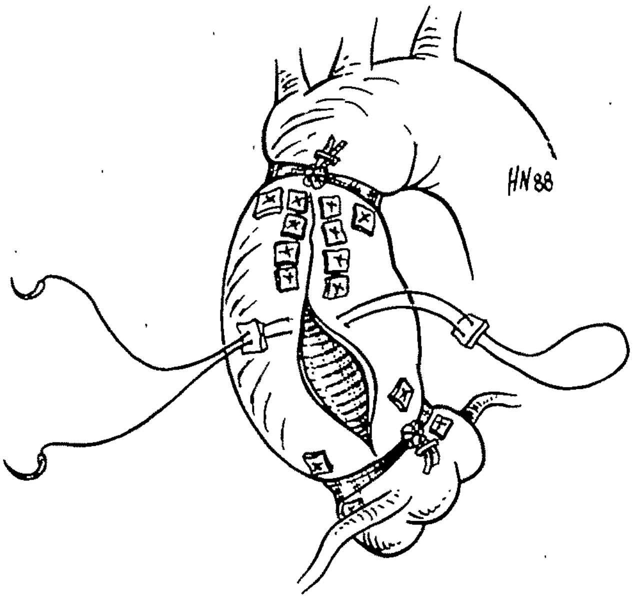

Fig 7. To avoid graft compression the aneurysm wall should not be completely closed until interstitial bleeding through the graft has ceased.

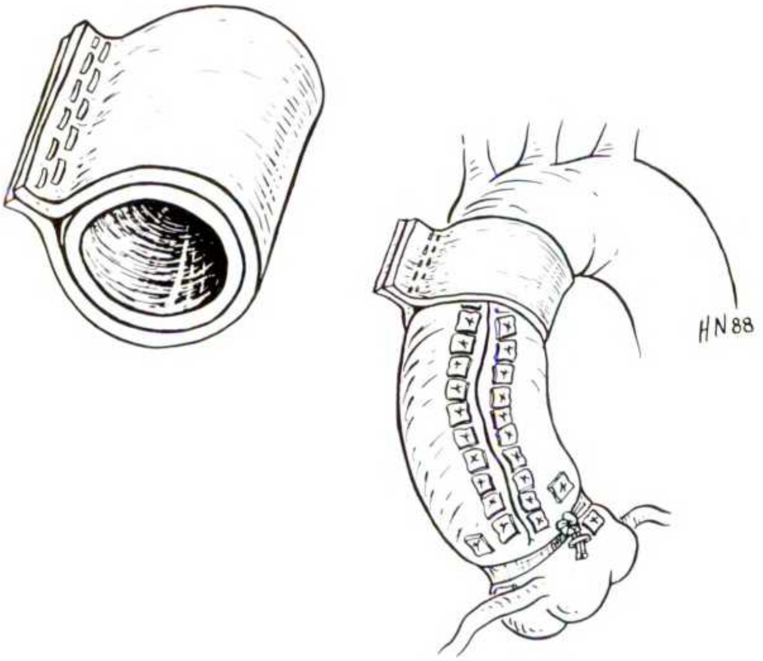

Fig 8. A 2-cm Teflon felt girdle fixed with a vascular stapler will relieve shearing stress on the aortic wall distal to the umbilical tape holding the distal spool. Pump flow is reduced as the stapler is fired to achieve a snug fit.

Results

Cardiopulmonary bypass time averaged 3.2 hours including a mean period of 35 minutes of circulatory arrest. On average the patients spent 5.9 hours in the operating room including anesthesia time. The mean hospital stay was 19 days including 1 patient who had a 5-week hospitalization because of respiratory difficulties secondary to a paralyzed left diaphragm.

The postoperative blood loss and transfusion requirements were minimal in all cases. Mean cumulative mediastinal shed blood was 123 mL by 12 hours after operation. Average transfusions were 4.7 units of packed red cells and 17.8 units of platelets for the entire hospitalization. Only 1 patient received fresh frozen plasma. The mean preoperative hematocrit was 0.40. The mean hematocrit at the time of discharge was 0.31.

There were no deaths, and morbidity was minimal. One patient, a pediatric surgeon, suffered a temporary loss of recent memory, but recovered fully and returned to his practice within 1 year. All patients were studied with postoperative echocardiography and dynamic computed tomography. One patient had 1 to 2+ aortic insufficiency but has remained in New York Heart Association class II on digoxin and furosemide. All other patients had minimal or no aortic regurgitation. One patient had evidence of an evolving inferior myocardial infarction before operation but had no serious myocardial dysfunction postoperatively. Two of the 4 patients had radiographic persistence of the arch and descending aortic false lumens all the way to the abdominal aorta. These have not enlarged radiographically and have been asymptomatic.

Comment

The surgeon operating on acute ascending dissection must be prepared to repair or replace the aortic root by a variety of methods because precise preoperative anatomical definition may not be possible. Olinger and associates [11] noted that the primary goals of the procedure in the setting of acute ascending dissection are (1) prevention of retrograde dissection and intrapericardial hemorrhage, (2) establishment of aortic valve competence, and (3) prevention of coronary ostial occlusion. Although control of the primary site of intimal disruption is desirable, it is not imperative. Several authors [12, 13] have noted that a majority of patients with ascending aortic dissection have asymptomatic late persistence of the distal false lumen after repair. This occurrence may be treated safely with β-blockade, antihypertensive therapy, and observation. As with acute primary dissection confined to the descending aorta, the indications for further surgical repair include visceral ischemia, late dilatation of the false channel, and documented redissection. In situations where the site of entry cannot be found in the ascending aorta the intraluminal technique can still be used to control the proximal dissection. In 1 of our patients the primary intimal tear was discovered in the transverse aorta above the distal spool of the intraluminal device. It was possible in this case to visualize and repair the rent in the lesser curvature of the aortic arch from within because of our use of deep hypothermic circulatory arrest. That patient underwent postoperative angiography at 1 year and was found to have no persistence of the false lumen in the aortic arch.

We have found that intraluminal graft repair with resuspension of the aortic valve using hypothermic circulatory arrest can safely achieve the goals of surgical therapy. The intraluminal "anastomosis" and inclusion technique affords several advantages over direct suturing methods. The use of an open technique during circulatory arrest allows thorough unimpeded evaluation of the entire ascending aorta and avoids clamp trauma to these notoriously delicate layers. The dissected aortic lumen is better controlled by not suturing the friable aortic wall directly. Hemorrhagic complications have not occurred even though profound hypothermic perfusion is used. Because large volumes of transfused components are not necessary the myocardial and infectious complications of this type of therapy for coagulopathy are avoided. When the aortic valve can be resuspended, there is no need for postoperative anticoagulation. The method is technically simpler than direct suture methods, which require reinforcement of suture lines with Teflon felt. Because of the relative simplicity of the technique cardiac ischemic time is reduced and myocardial injury and resulting low output postoperatively are lessened. The Meadox variable-length prosthesis has obviated the need for a large inventory of grafts in the operating room.

The limitations of this technique are related to the anatomical extent of the dissection and the premorbid anatomy of the ascending aorta and aortic valve. If there is clinically significant annuloaortic ectasia the resuspension will not restore competence. In Marfan's syndrome the sinus segment of the aorta should be replaced as serious late dilatation can result [14]. When acute dissection involves the sinus segment of the aorta total aortic root reconstruction is also necessary. In instances such as these we have performed a Bentall-Cabrol reconstruction [15] proximally. During the proximal repair the patient has been fully cooled to deep hypothermic levels so that a brief period of arrest may be used to perform the distal "anastamosis." We have found it useful in these cases to preclot the composite graft during placement of the aortic valve sutures by autoclaving the prosthesis in 5% albumin for 5 to 10 minutes. After the proximal repair is complete a mobile spool from an appropriately sized intraluminal graft is selected and fixed to the distal of the conduit. This is then used to create the distal "anastomosis." This technique was successfully used in 3 patients with acute dissection who were not acceptable candidates for aortic valve resuspension. All 3 patients survived, and there were no problems with bleeding. One patient who had a dissection extending into the right coronary ostium sustained a right ventricular myocardial infarct from intentional sacrifice of a nondominant right coronary artery, but recovered to New York Heart Association class I status.

Kouchoukos and co-workers [16] have suggested that inclusion techniques (hemostatic closure of the aneurysm wall over the completed reconstruction) increase the likelihood of graft compression and false aneurysm formation during composite graft replacement of the aortic root. This is particularly important if a modified intraluminal and composite valved conduit is used to reconstruct the aortic root because the coronary ostial anastomoses are at particular risk of pseudoaneurysm formation. We have not encountered pseudoaneurysm formation at "sutureless" anastomoses and suspect the likelihood of this occurring is substantially less than with hand-sewn anastomoses. Graft compression can be avoided by waiting until protamine sulfate has been administered and all interstitial graft oozing has ceased before closing the perianeurysmal space hemostatically.

We do not advocate the use of this technique if there is major dilatation of the transverse aorta. Although one may be able to fix the distal spool of the prosthesis securely, the size disparity that results will create a great deal of shearing force on the aortic wall distal to the circumferential tape. Further dilatation and possible disruption of the graft could result.

In summary, we have found that use of the variable-length intraluminal graft with hypothermic circulatory arrest for select cases of acute ascending aortic dissection is safe. The technique is applicable for ascending dissection when the aortic root is not chronically dilated and the aortic valve has normal anatomy. Bleeding and myocardial failure have not been factors in recovery after this technique. A modification of the technique can be used in combination with the Bentall-Cabrol procedure for the distal anastomosis. The device marketed by Meadox Medicals Inc has definitive advantages over the previously available fixed-length products and has allowed us to tailor the prosthesis to the operation.

We advocate the use of profound hypothermic arrest for virtually all operations on the acutely dissected ascending aorta. Neurological problems have not occurred and the absence of a clamp has allowed greater ability to evaluate and repair the dissection free of distortion and tension.

We gratefully acknowledge the assistance of Hugh Nachamie, who rendered the operative diagrams pictured here.

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Availability of Requests for Applications (RFA)

Division of Heart and Vascular Diseases
National Heart, Lung, and Blood Institute

Mechanisms of Damage Caused by Cardiopulmonary Bypass
NIH 90-HL-12-H
Application receipt date: December 3, 1990

The Devices and Technology Branch of the Division of Heart and Vascular Diseases, and the Blood Diseases Branch of the Division of Blood Diseases and Resources, announce the availability of a Request for Applications on the above subject. Applications are invited for support of basic research, studies with animal models, and clinical investigation targeted toward (1) obtaining a better understanding of the mechanisms of multisystem damage seen with cardiopulmonary bypass and the often associated technique of profoundly hypothermic total circulatory arrest, and the particular susceptibility to this damage of the very young and the elderly, and (2) developing methods of preventing or attenuating this damage.

Applicants are encouraged to develop their own approaches within their areas of expertise to investigating fundamental mechanisms of the complex humoral, cellular, and other responses to cardiopulmonary bypass; to developing reversible methods of preventing or minimizing these responses; or to exploring and testing in vivo newly developed methods of management.

Copies of the RFA may be obtained from:

Paul Didisheim, MD
Devices and Technology Branch
Division of Heart and Vascular Diseases
National Heart, Lung, and Blood Institute
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Facsimile: (301) 480-6282

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National Heart, Lung, and Blood Institute
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Intraoperative Bronchial Aspiration of Ruptured Pulmonary Hydatid Cysts

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Ruptured pulmonary hydatid cysts are seen clinically and radiologically as persistent cavitary lesions of the lung. Bronchi opening into the pericyst cavity allow for discharge of fluid matter but not the escape of solid remnants of the collapsed parasite. Operative manipulation of the involved lung in the course of surgical management of chronic ruptured pulmonary hydatids can force fragments of the laminated membrane or small daughter cysts into the bronchial tree. Such extruded solid fragments lodge in bronchi of the same or opposite lung with resulting acute obstruction of airways. Intraoperative bronchial aspiration of hydatid material was seen in 7 patients with ruptured hydatid cysts of the lung, either primary or secondary to transdiaphragmatic extension of liver hydatids. The first clinical sign can be unexplained difficulty in ventilation. Effective management consists of prompt exposure of the stem bronchus on the operative side, with bronchotomy for suction retrieval of escaped solid fragments of the parasite.

(Ann Thorac Surg 1990;50:631–6)

Hydatid cysts of the lung are uncommon in industrialized countries, but present a clinical problem of some magnitude in areas of the world endemic to echinococcal disease. Medical treatment has been reportedly successful in some cases of liver involvement [1–3], but operation remains the primary treatment modality for pulmonary echinococcal cysts. Lobectomy can be done. A conservative approach, however, consisting of removal of the parasite and suture closure of the bronchial openings is preferred, as there is primarily compression and no actual destruction of lung tissue [4]. Results are generally good as most patients are young, and *Echinococcus granulosus* disease has no systemic ill effects.

A special type of intraoperative complication was encountered in the course of surgical treatment of pulmonary hydatid cysts in an area of the world highly endemic to echinococcal disease: During operative manipulation of the affected lung, solid fragments of the parasite's previously ruptured laminated membrane escaped into the bronchial tree, resulting in acute bronchial obstruction. This report deals with 7 patients in whom such intraoperative bronchial aspiration of hydatid material occurred, and discusses a plan of management that evolved as a result of this experience.

Case Reports

Patient 1

A 42-year-old woman with a history of two previous laparotomies for hydatid cysts of the liver was seen with hemoptysis and consolidation of the right lower lung field. Bronchoscopy had been attempted elsewhere and the patient refused to undergo repeat bronchoscopy.

At operation the right lower lobe was found to be completely atelectatic, a hydatid cyst of the liver having penetrated the diaphragm to enter the substance of the right lower lobe, forming an irregular pericyst cavity about 7 cm in diameter. The cavity was opened and its content of mostly degenerated fragments of laminated membrane and a few small daughter cysts was evacuated. Two or three medium-sized bronchial openings entering the pericyst cavity were at first individually suture ligated, but as the lobe could not be made to expand, a lobectomy was carried out.

The anesthetist had difficulty in keeping the patient's airways open during lobectomy despite vigorous tracheal suctioning, and at the conclusion of the operation the patient's condition was found to be rapidly deteriorating. A portable chest roentgenogram showed complete atelectasis of the opposite lung (Fig 1). Bronchoscopy was attempted but cardiac arrest developed. She was rapidly reintubated and the left chest was speedily opened without further preparation.

Acting on the assumption that a piece of hydatid laminated membrane might have entered the left main bronchus during manipulation of the right lower lobe, we quickly opened the posterior hilum of the collapsed left lung and opened the left stem bronchus through a transverse incision. Fragments of hydatid cyst membrane and small daughter cysts almost filling the palm of a hand extruded or were extracted from within the bronchial lumen. Temporary digital occlusion of the bronchotomy allowed full expansion of the left lung, and normal heart beat resumed after a few manual cardiac compressions. The bronchotomy was closed and the operation terminated. Recovery was uneventful.

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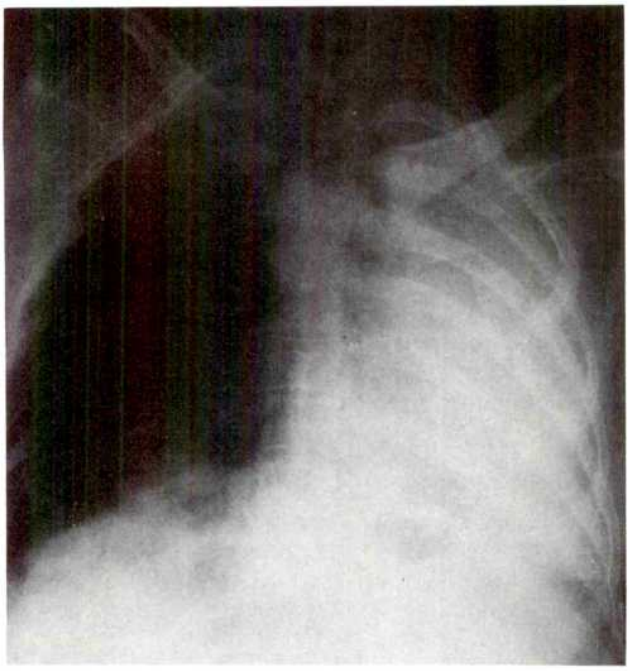

Fig 1. Portable chest roentgenogram of patient 1 taken immediately after right thoracotomy for ruptured hydatid cysts of the liver and right lower lobe, showing total atelectasis of the left lung secondary to aspiration of hydatid debris with occlusion of left main bronchus.

Patient 2

A 19-year-old man gave a 1-month history of occasional hemoptysis. Admission chest roentgenogram (Fig 2) showed a soft irregular density in the left lower lung field consistent with a previously ruptured hydatid cyst and a domelike elevation of the right hemidiaphragm suggested a coexisting large hydatid cyst of the liver.

The large liver cyst was first evacuated transdiaphragmatically through the right side of the chest. Six weeks later the left lung lesion was exposed through a left posterolateral thoracotomy and found to consist of a ruptured, tangerine-sized hydatid cyst within the substance of the basilar segments of the left lower lobe. Its presenting surface in the major fissure was incised and collapsed fragments of hydatid laminated membrane were evacuated from within a thick-walled, irregular, 4 × 6-cm pericyst cavity. At least two moderate-sized bronchial openings were found entering the pericyst cavity, and these were closed with fine silk sutures.

At this point it was noted that the grossly normal left upper lobe could not be made to expand. On the basis of the experience with the previous patient an aspiration-occlusion of the left upper lobe bronchus was suspected. The nearby left stem bronchus was exposed and opened transversely and small pieces of laminated membrane were retrieved on intrabronchial suctioning. There was no evidence of any membrane fragments having escaped into the opposite right lung.

Patient 3

A 52-year-old man with a history of multiple previous operations for bilateral lung and liver hydatid cysts was admitted because of intermittent hemoptysis. Chest roentgenogram showed an irregular density above the right hemidiaphragm, and with a presumptive diagnosis of ruptured hydatid cyst of the right lower lobe a thoracotomy was performed.

Dense adhesions encasing the right lower lobe and covering its diaphragmatic surface were taken down and the pathology was found to consist of two ruptured pericyst cavities, one in the liver and one in the lung, the two communicating with each other across the diaphragm. A considerable amount of degenerated gelatinous hydatid material was removed from both cavities and their uniting channel was divided and closed. At this point the anesthetist felt unable to inflate the patient's lungs, and vigorous endotracheal tube suctioning would not overcome a persistent airway obstruction.

The right main bronchus was quickly exposed in the posterior mediastinum and its membranous portion opened transversely about 2 cm beyond the carina. The lumen of the bronchus was empty, but when a No. 14 suction catheter with a single terminal hole was guided into the opposite bronchial tree five separate fragments of laminated membrane, each measuring about 1 × 2 cm in diameter, were extracted from the opposite bronchial tree. The bronchotomy was repaired and the right lower lobe pericyst cavity was managed by suture closure of its multiple bronchial openings.

Patient 4

A 42-year-old man had noted intermittent hemoptysis for 6 months, the chest roentgenogram showing an irregular lesion close to the hilum of the right lung. No pathology was seen on bronchoscopy. At thoracotomy the right middle lobe was found to be firmly atelectatic and surrounded by soft pleural adhesions. These were freed and the lesion was incised, exposing an irregular, thick-walled

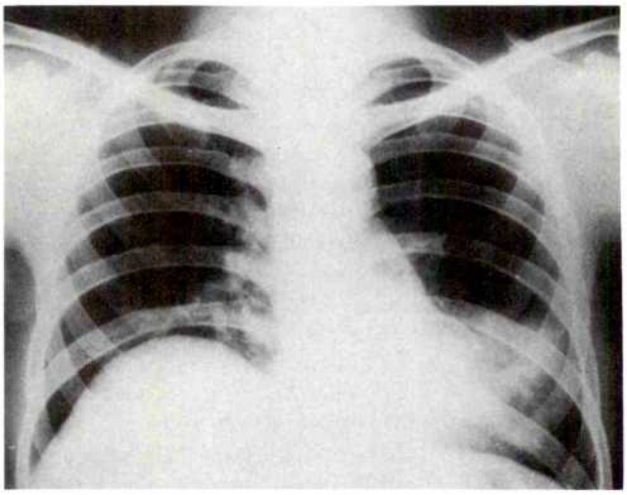

Fig 2. Posteroanterior chest roentgenogram of patient 2. The irregular soft density in the left lower lung field and the smooth domelike elevation of the right hemidiaphragm, taken together and in an endemic setting, are highly suggestive of synchronous pulmonary and hepatic hydatid cysts.

pericyst cavity about 4 cm in diameter. This contained some amorphous fibrinous and purulent material but no fragments of laminated membrane as such. Three separate medium-sized bronchial openings that entered the pericyst cavity were sutured, and the operation was terminated uneventfully.

A portable chest roentgenogram taken 3 hours postoperatively showed general haziness and multiple irregular opacification of the opposite lung. With intensive chest physiotherapy the patient coughed up two separate fragments of laminated membrane and repeat chest roentgenogram showed clearing of the left lung.

Patient 5

A 56-year-old woman gave a 2-month history of epigastric and right chest pain, with chronic cough and hemoptysis during the preceding 2 years. Chest roentgenogram showed multiple ruptured cysts and irregular nonspecific densities on the right side consistent with previously ruptured hydatid cysts. Liver scan showed multiple small and large space-occupying lesions, presumably also of hydatid nature. The operative plan was to evacuate the right lung cysts and transdiaphragmatically deal with as many liver cysts as possible.

A right seventh interspace posterolateral thoracotomy led promptly into a large irregular cavity within the substance of the right lower lobe as the pleural space had become totally obliterated by dense adhesions from previous operations. A number of bronchial openings were visible at the bottom of the cavity. Shortly after evacuating the contents of the pericyst cavity, the anesthetist felt that the patient could no longer be adequately ventilated and a few moments later announced that the patient was in cardiac arrest.

Access to the right main bronchus was difficult because of the dense pleural adhesions in the posterior mediastinum. Eventually the trachea was reached and opened just above the carina. The opposite bronchial tree was quickly entered with a large-bore suction catheter and a considerable amount of gelatinous fragments of laminated membrane was aspirated. The left lung could now be expanded but cardiac resuscitation was unsuccessful and the patient died on the operating table.

Patient 6

The chest roentgenogram of a 22-year-old woman with cough and hemoptysis of about 6 months' duration showed a round homogenous lesion of the right lower lobe. There was also an irregular, faintly calcified lesion in the right lobe of the liver beneath the diaphragm that, in retrospect, should have been diagnosed as a previously ruptured and partially calcified hydatid cyst of the liver.

The pathology was found to be a chronically ruptured hydatid cyst of the liver that had penetrated the diaphragm to invade the right lower lobe. Contents of both liver and lung pericyst cavities, consisting of degenerated fragments of laminated membrane and a few small daughter cysts, were completely evacuated and two bronchial openings were suture closed. Because the right lower lobe would not expand a lobectomy was carried out uneventfully.

A routine postoperative portable chest roentgenogram showed expansion of the remaining right upper lobe, but there was total opacification of the opposite left lung. Aspiration of hydatid material was suspected and the patient underwent bronchoscopy. Fragments of laminated membrane and a few small and medium-sized daughter cysts were patiently removed with biopsy forceps and by suctioning; repeat chest roentgenogram showed complete expansion of the left lung.

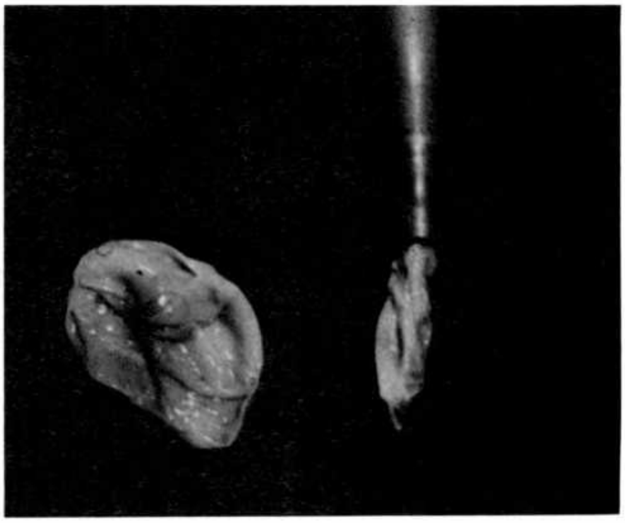

Fig 3. (Patient 7.) The single ruptured hydatid cyst on the left had already been surgically removed from the lingula, when a separate unsuspected ruptured hydatid cyst of the left lower lobe escaped into the bronchial tree to reach and occlude the tip of the endotracheal tube.

Patient 7

A 2.5-year-old girl—the youngest patient with hydatid disease seen by us—had multiple bilateral lung cysts. The right lung was first operated on and four cysts were removed. Three weeks later left thoracotomy was performed, and what was palpated to be a single cyst in the lingula was extracted, and two small bronchial holes entering its soft-walled pericyst cavity were sutured. At this time the anesthetist noted that the left lung would not deflate, and moments later the lung could not be inflated at all. The patient was rapidly turned over to the supine position, and the endotracheal tube was removed and replaced with a new one. Bradycardia ensued but reverted on reestablishment of a satisfactory airway. The tip of the removed endotracheal tube was seen to have become blocked by a single collapsed hydatid cyst (Fig 3). The source of this second cyst was searched for and found to be an empty pericyst cavity within the substance of the lower lobe. This had been missed as it had apparently ruptured shortly before or perhaps during the operation, and was thus no longer palpable as a discrete lesion.

Comment

Viable intact echinococcal pulmonary cysts continue to enlarge until they rupture or are removed. Accordingly,

Table 1. Clinicopathological Summary of Patients With Intraoperative Bronchial Aspiration of Hydatid Material

Patient No.	Age (y)	Sex	Location of Cyst(s)	Procedure Carried Out	Signs of Bronchial Obstruction	Management	Results
1	42	F	Right lower lobe and liver	Right lower lobectomy, evacuation of liver cyst	Progressive resistance to ventilation, total atelectasis opposite lung, cardiac arrest	Rapid opposite side thoracotomy, stem bronchus opened	Large amount of membrane fragments recovered, patient did well
2	19	M	Left lower lobe, separate liver cyst	Evacuation of lower lobe pericyst cavity	Left upper lobe would not expand	Left stem bronchus opened	Small pieces of membrane extracted, left upper lobe expanded
3	52	M	Right lower lobe cyst communicating with liver cyst	Evacuation of lung and liver pericyst cavities	Inability of anesthetist to inflate lungs	Right stem bronchus opened	Five separate fragments of membrane retrieved, lungs inflated
4	42	M	Right middle lobe	Evacuation of middle lobe pericyst cavity	Postoperative patchy atelectasis of opposite lung	Vigorous chest physiotherapy	Fragments of laminated membrane expectorated, atelectasis cleared
5	56	F	Multiple cysts of right lung and liver	Evacuation of right lung cyst attempted	Rapidly progressive ventilatory obstruction, cardiac arrest	Exposure of stem bronchus impeded by dense adhesions	Large amounts of membrane fragments recovered, patient died
6	22	F	Right lower lobe and liver	Right lower lobectomy, evacuation of liver pericyst cavity	Postoperative total atelectasis of opposite lung	Emergency bronchoscopy	Bronchoscopic extraction of membrane fragments, left lung expanded
7	2.5	F	Lingula and left lower lobe	Evacuation of pericyst cavity of lingula	Inability to deflate, then inflate lungs	Rapid replacement of blocked endotracheal tube	Ventilation reestablished, patient did well

all hydatid cysts of the lung are seen clinically and radiologically as either intact or ruptured cysts. A ruptured cyst will no longer grow in size, but is transformed into a chronic abscess cavity of a special type: Bronchial holes opening into the thick-walled pericyst cavity allow for drainage of hydatid fluid and accumulating secretions, but their orifices are generally too small to permit discharge of fragments of the collapsed laminated membrane or any daughter cysts that may have formed within the cavity. There is no actual tissue destruction, but the festering cavitary lesion can only be managed surgically by either lobectomy or removal of retained hydatid debris and suture closure of the draining bronchial openings. And because, unlike the situation for intact hydatid cysts, no viable scoleces are present in these chronically ruptured pericyst cavities, there is usually no need for the use of scolicidal agent.

Very small pieces of broken-up laminated membrane and even collapsed tiny daughter cysts do escape spontaneously at times into the bronchial tree, as patients occasionally give a history of having coughed up bits of grapeskin-like material. Although such spontaneous expectoration of hydatid debris can theoretically end in complete cure, there is the danger of asphyxiation if a sizeable piece of laminated membrane becomes impacted in the glottis, as was pointed out some 60 years ago by Harold Dew of Australia [5].

The present experience documents an iatrogenic escape of hydatid material from the pericyst cavity with resulting bronchial aspiration-occlusion in 7 patients whose clinicopathological state is summarized in Table 1.

In all but 1 patient, the hydatid debris escaped from within the pericyst cavity of long-standing and previously ruptured cysts; in only 1 patient (patient 7) did an intact cyst rupture during or just before the operation. It did not matter whether lobectomy or simple evacuation of the pericyst cavity had been carried out, as the time sequence of events left little doubt that intraoperative manipulation had led to intrabronchial escape of the debris. The actual amount of escaping solid hydatid material varied from a sizeable aggregate of fragmented pieces of laminated membrane and small daughter cysts to a single medium-sized collapsed cyst. In 4 patients (patients 1, 3, 5, and 6) the source of the debris was clearly a long-standing primary cyst of the liver that had penetrated the diaphragm to establish secondary dwelling in the right lower lobe, the multivesicular hepatic cyst thereafter feeding small daughter cysts into the lung cyst.

The final destination of escaping hydatid material also varied. In patients 1 and 5 the main opposite bronchus had become plugged, whereas only an ipsilateral lobar bronchus became occluded in patient 2. The single collapsed cyst of patient 7 caused at first partial then total occlusion of the tip of the endotracheal tube. Patient 5 died on the operating table, perhaps not so much because of the degree of acute bronchial obstruction as because of the delay in relieving it.

The 7 patients presented were encountered over a period of 5 years in a highly endemic area during which some 85 patients with intact and ruptured hydatid cysts of the lung were operated on, or personally supervised, by the principal author (F.S.). Not all primary lung cysts were of the chronically ruptured variety, and 9 patients had a primary liver hydatid that had crossed the diaphragm to enter the lung. The overall incidence of the complication described would, accordingly, be not more than about 5% in any large series.

A correct preoperative diagnosis of ruptured pulmonary hydatid cysts is based primarily on clinical experience with this disease in an endemic setting. Ruptured pulmonary cysts, unlike intact ones, do not show characteristic radiologic signs. Bronchoscopy does not establish the diagnosis, and bronchoscopic exploration of a pericyst cavity is not practicable.

The correct diagnosis of intraoperative escape of hydatid material may also not be made right away, as clinical signs of bronchial occlusion on the same or opposite side may not be very striking. Again, familiarity with the basic pathology involved is essential. Any unaccountable changes in the mechanics of ventilation should alert the operating team to the possibility of bronchial aspiration-occlusion. Fiberoptic transendotracheal bronchoscopy may or may not establish the diagnosis, but it certainly cannot effect prompt relief of the obstruction.

Prevention of intraoperative bronchial aspiration of hydatid material, assuming the surgeon has recognized the pulmonary lesion as a previously ruptured hydatid cyst, rests on (1) avoiding undue pressure on the involved lung and (2) having the stem bronchus exposed should the need for prompt bronchotomy arise. Routine use of a double-lumen endotracheal tube might appear a proper step in the prevention or management of intraoperative bronchial aspiration of hydatid material. Such tubes would prevent entry of escaping fragments of laminated membrane into the bronchial system of the opposite lung, but airway occlusion on the operative side can still occur, and retrieval of hydatid fragments from within the narrower lumen of these tubes would be even more difficult than through standard single-lumen endotracheal tubes.

Management of the described intraoperative complication must in any case be prompt even if the underlying cause is not immediately apparent. The anesthetist usually reflexively attempts suctioning through the endotracheal tube with the largest catheter available. The soft and gelatinous physical characteristics of the hydatid material may not allow a rapid and complete suction retrieval. Suctioning has furthermore the undesirable effect of impacting solid hydatid membrane fragments further down the bronchus. Removal and reinsertion of the endotracheal tube in an adult patient in the lateral decubitus position is neither easy nor convenient, bronchoscopy being less so.

On the basis of the experience described, the suggested approach to the complication described is, first, a prompt recognition of the underlying cause, followed by rapid and direct entry into the largest portion of the bronchial system. The membranous portion of the mobilized stem bronchus is opened transversely on the operative side. Next a short, end-hole plastic catheter of the largest size that will easily enter the main bronchus is used for general toilet of the bronchial tree on the opposite and same side. The whole maneuver will not take more than 20 to 30 seconds if the stem bronchus has already been exposed in the posterior hilum and separate suction equipment is on standby. Full and free expansion of the lungs while the bronchotomy is temporarily occluded with a fingertip is the best assurance of having evacuated any aspirated laminated membrane fragments while they are still floating within the bronchial tree. The bronchotomy is closed with a few fine silk sutures and the offending pericyst cavity is managed along the lines described.

The above approach has become a routine measure for us for cases of chronically ruptured hydatid cysts of the lung, especially when there is an associated multivesicular hydatid cyst of the liver penetrating the diaphragm. Finally, it is suggested that when operating on ruptured as well as intact hydatid cysts of the lung, the anesthetist ventilate the patient manually throughout the procedure. Not only will this create positive pressure within the bronchial system counteracting the surgeon's external pressure on the lung, but the anesthetist's sensation of minimal resistance to inflation may be the first clue to acute bronchial aspiration-occlusion. An early postoperative chest roentgenogram should be routine to detect an unsuspected complication of the nature described.

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INVITED COMMENTARY

Bronchotomy followed by direct retrieval is an effective rescue procedure in case of aspiration of fluid matter or daughter cysts during operation for hydatid cysts. However, this complication can usually be avoided by applying the following guidelines: (1) Intubate the lungs separately. This prevents flooding of the contralateral lung and allows the anesthesist to aspirate any cyst fluid matter that may enter the bronchus. (2) If lobectomy is performed, check patency of the remaining lobe(s) before actuating the stapler used to make the bronchial suture. If the lobe fails to inflate, remove the stapler and open the bronchus distal to the presumed point of section. (3) In case of ruptured cysts, carry out fiberscopy immediately after operation while the patient is still on the table.

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Value of Transesophageal Echocardiography During Repair of Congenital Heart Defects

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Two-dimensional transesophageal color Doppler echocardiography was employed intraoperatively in 30 children undergoing repair of a variety of simple and complex cardiac malformations. There were 16 female and 14 male patients, with a mean age of 9 ± 3 years (range, 4 to 13 years) and a mean weight of 31 ± 9 kg (range, 16 to 50 kg), 16 children weighing less than 30 kg. A standard, commercially available transesophageal echocardiography probe (5 MHz, 64 elements) was used in all patients without complications. Transesophageal echocardiography proved helpful in selecting the surgical approach, in assessing the adequacy of surgical repair, in detecting residual intracardiac shunts, and in allowing uninterrupted monitoring of ventricular performance throughout the procedure. Our initial experience suggests that transesophageal echocardiography is a valuable tool to be used in children with congenital cardiac malformations, particularly in those requiring complex intracardiac procedures. The amount of information obtained by the surgeon should favor the routine use of transesophageal echocardiography during open heart procedures and stimulate the development of probes to be safely used even in infants and newborns.

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Transesophageal echocardiography (TEE) is currently gaining increasing popularity as a means of continuous cardiac imaging during open heart operations [1, 2]; its ability to monitor left ventricular function and wall motion abnormalities and to assess the adequacy of surgical repair is well defined in adult patients [3, 4].

With the recent introduction of color-flow Doppler technology in transesophageal imaging, TEE has become even more suitable for intraoperative application and its indications are extended to all situations in which detection of abnormal intracardiac blood flow is needed [5]. For this reason patients with congenital heart defects should benefit most from the intraoperative use of TEE. Experience in this field, however, is at present very limited [6, 7], mainly because pediatric-size probes were not commercially available until very recently, while epicardial color flow imaging has received increasing acceptance for intraoperative application in children [8]. The latter, however, presents many limitations, such as the need for temporary discontinuation of the operative procedure, impossibility of continuous imaging, poor surface contact, heart compression with potential rhythm disturbances, and, above all, limited time allowed for evaluation [9].

We present here our initial experience with two-dimensional (2-D) color Doppler TEE used intraoperatively in a series of children undergoing repair of various congenital heart malformations. This report focuses on the technical feasibility of 2-D TEE and its potential advantages for the cardiac surgeon.

Material and Methods

Patient Population

During the interval October 1988 to September 1989, 2-D TEE was used intraoperatively in 30 children undergoing surgical repair of various congenital heart defects. The criteria to be enrolled in the study were a body weight preferably above 20 kg and absence of any documented pathology of the upper gastrointestinal tract. There were 16 female and 14 male patients, with a mean age at operation of 9 ± 3 years (range, 4 to 13 years) and a mean weight of 31 ± 9 kg (range, 16 to 50 kg), 16 weighing less than 30 kg. Twenty-seven patients had primary repair of various intracardiac malformations, 1 patient had reoperation for residual pulmonary stenosis and ventricular septal defect (VSD) due to patch detachment after correction of tetralogy of Fallot, 1 had reoperation because of subaortic stenosis after repair of interrupted aortic arch and VSD, and 1 had an open chest biopsy of a left ventricular hemangioma (Table 1).

The preoperative diagnosis was made with standard 2-D echocardiographic and hemodynamic investigations in 13 and by 2-D echocardiography alone in 17. With the exception of the patient undergoing left ventricular biopsy all children were operated on with standard cardiopulmonary bypass and moderate systemic hypothermia. Myocardial protection was achieved in all cases with the use of cold potassium cardioplegia combined with topical cool-

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Table 1. Preoperative Diagnosis and Surgical Procedures

Disease	No. of Patients	Operation
Ostium II ASD	5	ASD patch closure
+ PAPVC	1	ASD patch closure, intraatrial tunnel
+PS	1	ASD patch closure, pulmonary valvotomy
Ostium I ASD	5	ASD patch closure, mitral valvoplasty (3)
Fibrous SAS	4	Blunt resection, septal myotomy (2)
VSD	1	VSD patch closure
+PS	2	VSD patch closure, pulmonary valvotomy (1), right ventricular muscle band resection (1)
+AI	2	VSD patch closure, aortic valvoplasty
Aortic stenosis	1	Commissurotomy and valvoplasty
TOF	1	TOF repair with transannular patch
+CAVC	1	TOF repair with transannular patch, CAVC repair with double patch
TGA, VSD, PS	1	Rastelli procedure with aortic homograft
Single ventricle, CAVC	1	Modified Fontan, atrial partitioning
Ebstein's anomaly TV[a]	1	Tricuspid valve replacement
VSD patch detachment, PS[b]	1	VSD closure, transannular patch
Fibrous SAS, AI[c]	1	Blunt resection
LV hemangioma	1	Left ventricular biopsy

[a] Underwent successful orthotopic heart transplantation because of biventricular failure after 20 hours of mechanical support. [b] Reoperation after previous repair of TOF. [c] Reoperation after previous repair of interrupted aortic arch and VSD closure.

AI = aortic incompetence; ASD = atrial septal defect; CAVC = complete atrioventricular canal; LV = left ventricular; PAPVC = partial anomalous pulmonary venous connection; PS = pulmonary stenosis; SAS = subaortic stenosis; TGA = transposition of great arteries; TOF = tetralogy of Fallot; TV = tricuspid valve; VSD = ventricular septal defect.

ing. Details of the surgical techniques employed are summarized in Table 1.

Technique of Two-Dimensional Transesophageal Echocardiography

A 64-element, 5-MHz, commercially available phased array transducer (HP 21362 A, Hewlett-Packard Co, Andover, MA) interfaced with a Hewlett-Packard ultrasonograph (HP 77020 AC) was used in all cases. This system allows color-flow Doppler examination as well as high-resolution 2-D imaging.

Insertion of the probe was performed in the anesthetized patient after endotracheal intubation, either blindly or under direct visualization of the pharynx with the neck moderately flexed. Advancement of the generously lubricated probe was accomplished very gently in the flexible mode with the control unlocked. Tilting of the transducer tip in both the anteroposterior and lateral planes was intentionally avoided in patients weighing less than 30 kg.

Transesophageal echocardiographic monitoring was started before skin incision and continued throughout the entire procedure, being temporarily suspended during cardiopulmonary bypass to minimize the risk of esophageal damage due to probe heating and temperature gradient during this period. At the end of each procedure, just before transfer of the patient from the operating room to the intensive care unit, the probe was removed. When indicated, 2-D TEE studies were also performed in the postoperative period while the patient was still intubated.

Cardiac views were obtained from both the esophagus and stomach as described by Seward and associates [10]. Longitudinal four-chamber or four-chamber and left ventricular outflow tract views were mainly used; additional views such as basal short-axis, transgastric short-axis, and transgastric four-chamber were also obtained when considered of particular interest for the study. For color-flow mapping long-axis views were almost exclusively used, as blood flow must be parallel to the Doppler source for optimal signal processing.

Results

Surgical Results

There were two hospital deaths (less than 30 days). One 12-year-old boy died of septic shock 4 days after total repair of tetralogy of Fallot and complete atrioventricular canal, and a 12-year-old girl died of heart failure on postoperative day 1 after a modified Fontan procedure with atrial partitioning for single ventricle and complete atrioventricular canal. Finally, a 12-year-old boy showed severe biventricular failure after tricuspid valve replacement for Ebstein's anomaly; he was supported with extracorporeal circulation for 20 hours before undergoing successful orthotopic heart transplantation. No major postoperative complications were observed in the remaining patients.

Technical Aspects of Transesophageal Echocardiography

Two-dimensional transesophageal echocardiography monitoring was performed 32 times, once in 28 patients and twice in 2. In the latter it was repeated in the intensive care unit within 24 hours after operation: in the patient with low output state after a modified Fontan procedure, to assess left ventricular performance, and in the patient with tetralogy of Fallot and complete atrioventricular canal repair to verify the effect of vasodilator administration on the degree of residual mitral incompetence.

The probe was successfully inserted without problems in all cases and was left in place for the entire surgical procedure in all but 1. In this patient, the smallest of our series (a 6-year-old, 16-kg boy), the probe was withdrawn during the period of cardiopulmonary bypass. The quality of 2-D TEE was considered satisfactory in all cases,

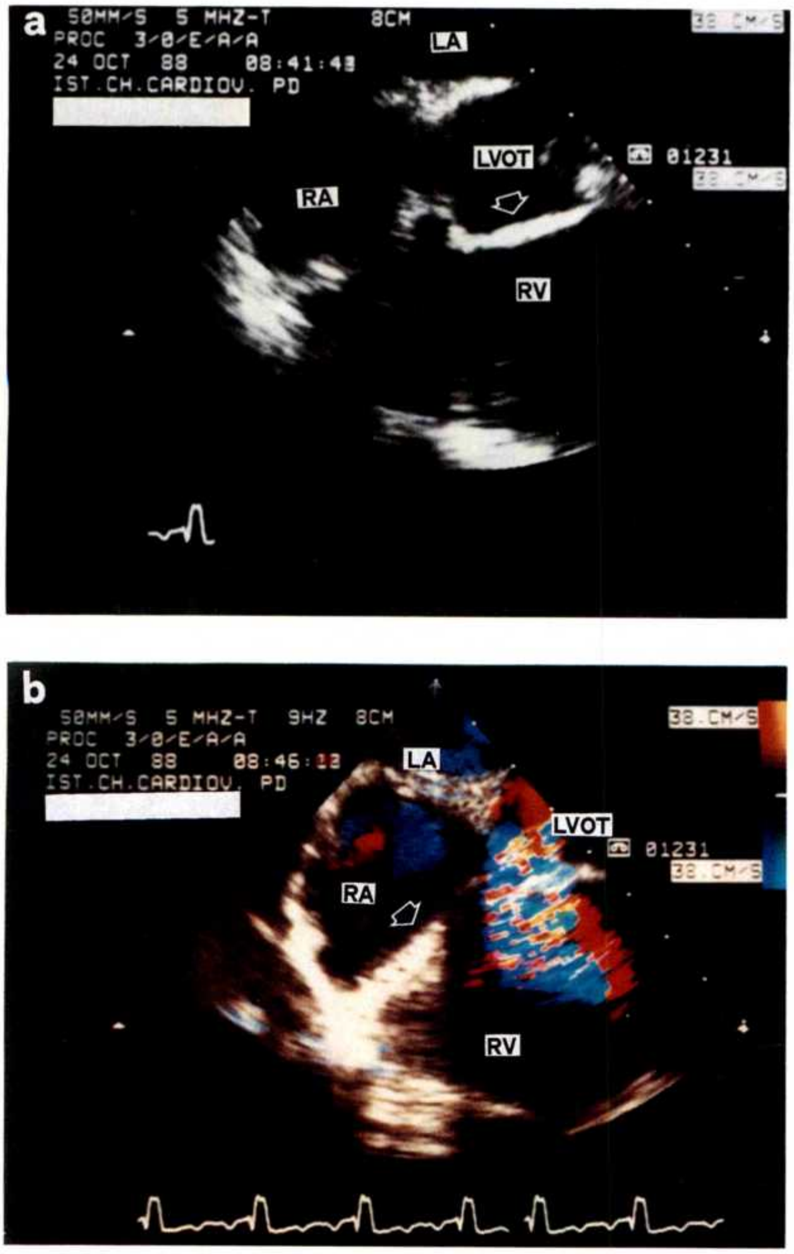

Fig 1. (a) Transesophageal two-dimensional modified long-axis view in a 5-year-old, 21-kg boy undergoing reoperation for ventricular patch detachment (arrow) *after correction of tetralogy of Fallot. (b) Two-dimensional color-flow Doppler echocardiography after slight rotation of the probe detecting a mosaic jet from the angle between the left ventricular outflow tract* (LVOT) *and the tricuspid plane* (arrow). (LA = *left atrium;* RA = *right atrium;* RV = *right ventricle.)*

including smaller patients (less than 30 kg in weight) in whom probe manipulation was intentionally avoided.

The addition of color-flow mapping, performed at a mean pulse repetition frequency of 3 to 4 KHz, was considered to represent a substantial improvement to the examination in all cases. Nevertheless, in some instances "ghost" color signals were derived from fast-moving cardiac structures instead of only from the bloodstream. This phenomenon was observed particularly when the heart rate exceeded 140 beats/min, as occurred in some of our children during weaning from cardiopulmonary bypass; in such cases time was allowed for rhythm stabilization while evaluation of color data was in the meantime performed through frame by frame analysis.

Atrial Septum

Both atria and the interatrial septum were always well visualized by a modified long-axis view, and adequacy of surgical repair after simple atrial septal defect patch closure was confirmed in all cases. Useful information also was provided by TEE, with the adjunct of color-flow Doppler, when an atrial septal defect was part of a more complex intracardiac malformation. In fact, in 1 patient, who showed signs of marked arterial desaturation immediately after a modified Fontan procedure for single ventricle and complete atrioventricular canal, a partial detachment of the pericardial baffle used for atrial septation was detected, thus allowing immediate repair of this complication; in this particular patient, however, the atriopul-

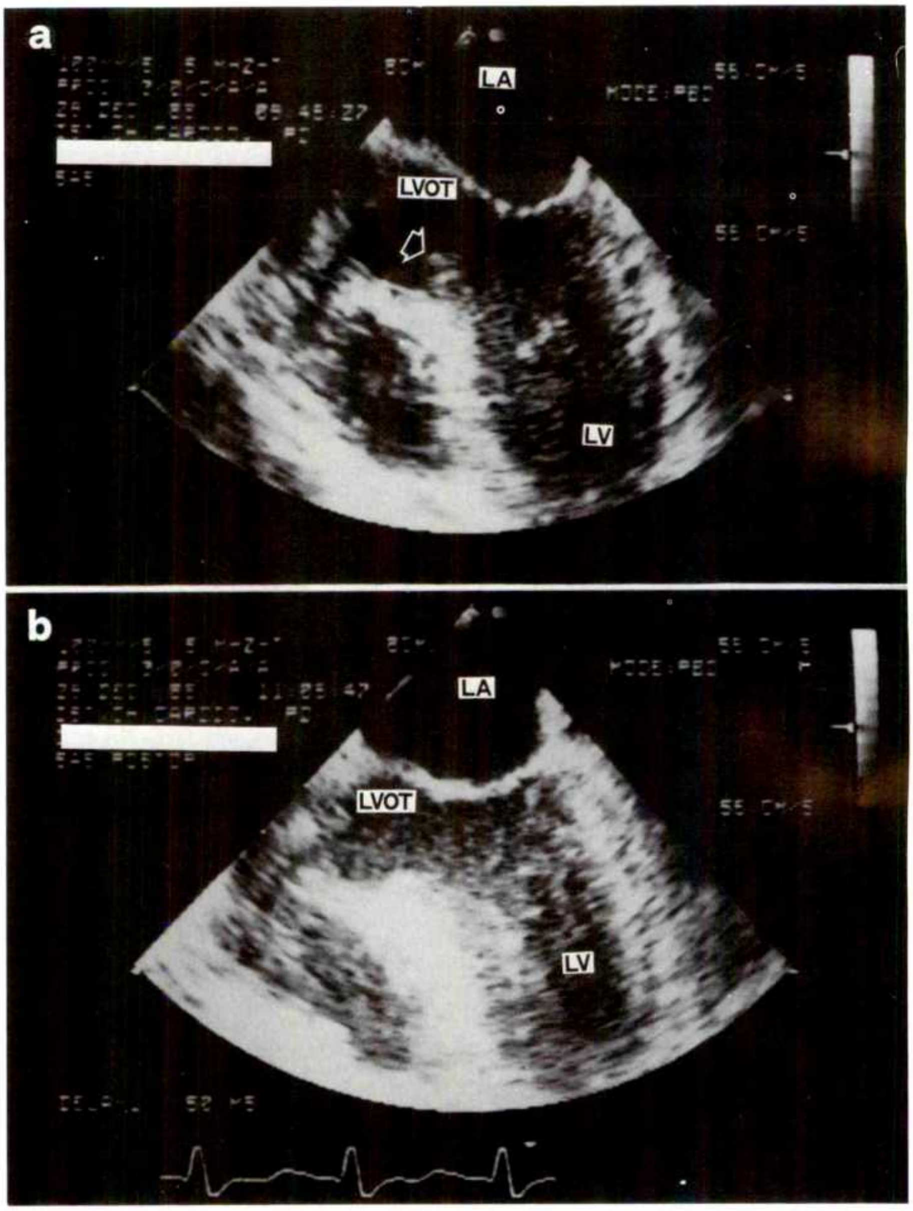

Fig 2. (a) Preoperative transesophageal two-dimensional long-axis view of the left ventricular outflow tract (LVOT) *in a 10-year-old, 37-kg boy showing a subaortic ridge* (arrow). *(b) Postoperative control in the same view demonstrating complete resection of the ridge.* (LA *= left atrium;* LV *= left ventricle.)*

monary anastomosis could not be effectively visualized. In another patient, in whom preoperative catheterization had shown a VSD and raised the suspicion of an atrial septal defect on the basis of oxymetric data, intraoperative TEE showed an intact interatrial septum; this finding was confirmed at direct inspection and the high oxygen saturation was shown to be caused by the combination of ventricular shunting and tricuspid regurgitation secondary to valve dysplasia.

Ventricular Septum

The ventricular septum was best visualized with a standard long-axis view. In all patients with perimembranous and malalignment VSD TEE was helpful to assess the adequacy of operation and to exclude any residual interventricular shunt. Particularly, in 1 child undergoing reoperation after tetralogy of Fallot repair it allowed exact identification of the site of patch detachment, not clearly shown at angiography (Fig 1).

Left Ventricular Outflow Tract and Aortic Valve

Both left ventricular outflow tract and aortic valve were always well identified through a long-axis view; in addition a basal short-axis view was also helpful in imaging the aortic valve. In patients with subaortic stenosis TEE allowed us to immediately verify the complete resection of the subaortic membrane and to exclude any iatrogenic complication such as a VSD or aortic regurgitation (Fig 2). Both aortic valve and left ventricular outflow tract were easily examined also in patients with aortic incompetence or stenosis to assess the early result of valve repair.

Pulmonary Artery

When needed, the main pulmonary trunk and right pulmonary artery were well visualized through a basal

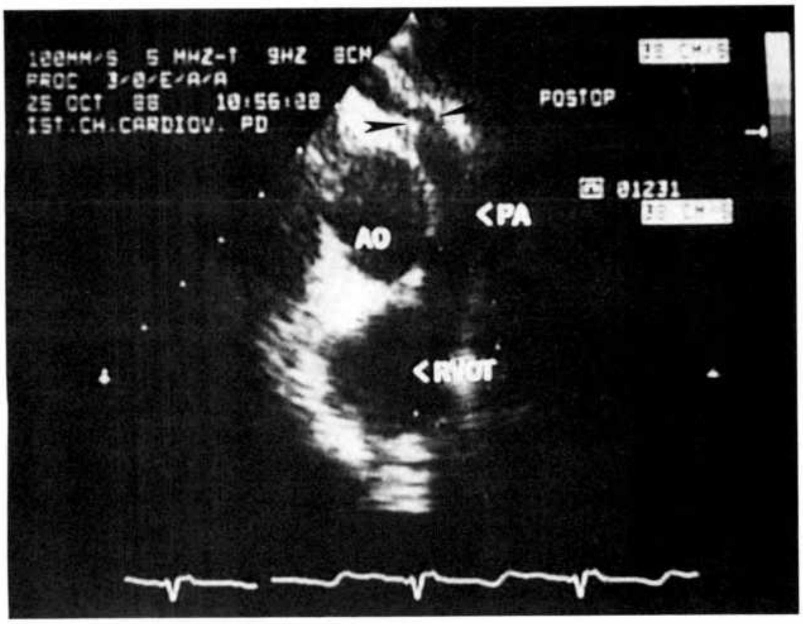

Fig 3. Transesophageal two-dimensional basal short-axis view after closure of a ventricular septal defect in a 7-year-old, 24-kg boy showing stenosis of the origin of the right pulmonary artery (arrowheads). (AO = aorta; PA = main pulmonary artery; RVOT = right ventricular outflow tract.)

short-axis view. In 1 patient stenosis of the right pulmonary artery could be demonstrated after VSD patch closure even though intraoperative measurements failed to reveal any clinically significant pressure gradient (Fig 3). However, in the child who had a Rastelli procedure for transposition of the great arteries the right ventricular to pulmonary artery conduit could not be visualized, nor could the right ventricular outflow tract and pulmonary valve in many patients.

Atrioventricular Valves

Both atrioventricular valves were always well identified in a long-axis, four-chamber view. Transesophageal echocardiography was of particular value in the evaluation of mitral anatomy and dynamics because of the favorable position of this valve with regard to the esophageal window; consequently, immediate evaluation of the adequacy of mitral valvoplasty was always possible. Furthermore, in the patient with tetralogy of Fallot and complete atrioventricular canal, TEE helped to assess the degree of residual mitral incompetence after repair of a severely dysplastic mitral component and to monitor the effect of vasodilator therapy in weaning the patient from cardiopulmonary bypass (Fig 4).

Cardiac Performance

Variations of cardiac function due to hypovolemia or hypocontractility were instantaneously recognized by TEE in all patients, allowing us to select the most proper treatment in each situation. However, quantitative analysis was often impractical mainly because of the hemodynamic instability of most patients with complex intracardiac repair early after cardiopulmonary bypass.

Comment

The clinical experience with the intraoperative use of TEE in children so far has been very limited. Recently, Cyran and co-workers [6] demonstrated the efficacy of this technique in children undergoing operation for congenital heart disease. They employed a 32-element, 3.5-MHz transducer mounted on a gastroscope with an 11-mm external diameter shaft and a 15-mm external diameter tip in all patients except 1, in whom they used a smaller and more technologically advanced second-generation probe. They demonstrated the feasibility of TEE in patients older than 7 years and the great potential of this technique in assessing the adequacy of surgical repair of complex cardiac lesions.

In all patients in our series we used a second-generation TEE imaging system that has a higher number of elements, 64, with an emission frequency of 5 MHz and smaller dimensions (10-mm external diameter shaft and 12 × 14-mm external dimension tip) and incorporates color-flow Doppler echocardiography. These features have further enhanced the utility of intraoperative TEE in pediatric patients undergoing open heart operations by allowing a higher resolution in the near field and, what is more important, a better understanding of normal and abnormal intracardiac blood flow before and after correction.

Transesophageal echocardiography was employed in 30 children between 4 and 13 years of age who had a mean weight of 31 ± 9 kg and a variety of simple and complex cardiac malformations. We were able to study children with a body weight as low as 16 kg by employing probes of smaller dimensions when compared with those used by others [6]. Although subsequent endoscopic examination was not performed in any patient in this series, no apparent complications related to probe insertion were observed, most likely because some technical guidelines were routinely followed. These included generous lubrication of the probe, moderate nuchal flexion, direct laryngoscopic visualization in the presence of even minimal resistance to probe advancement into the pharynx, and

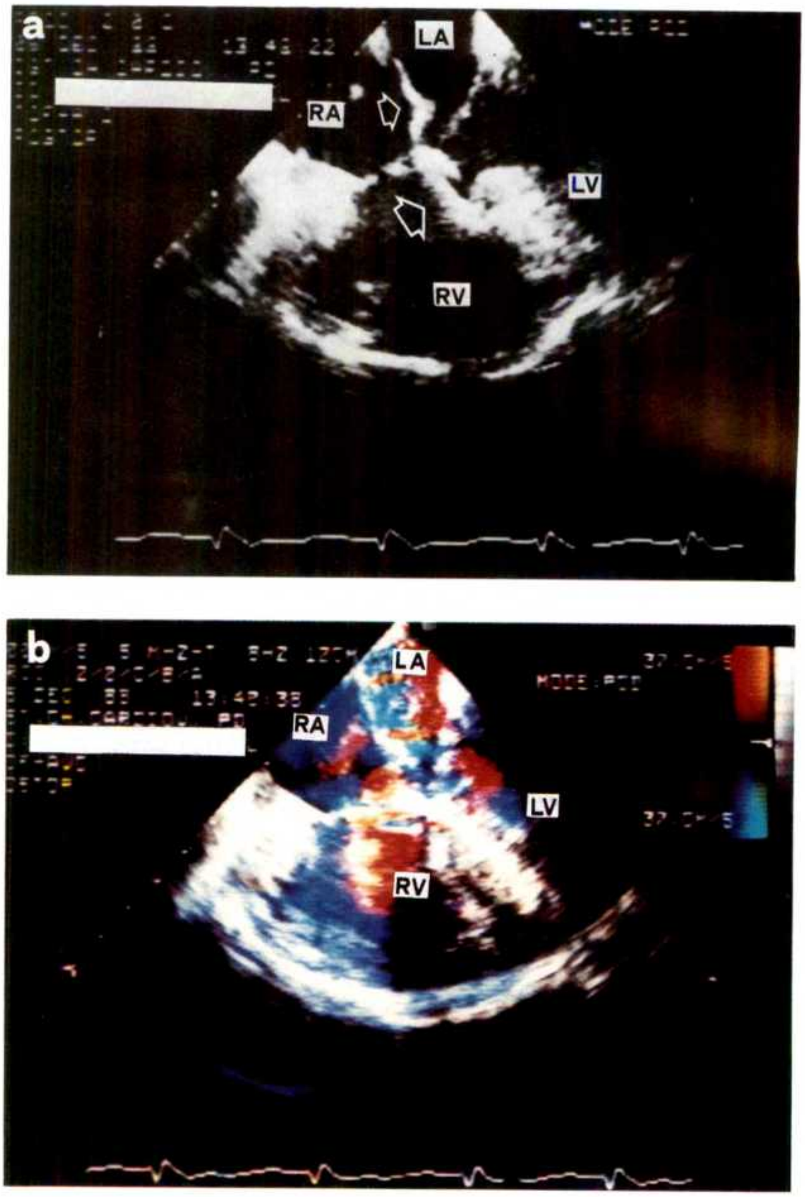

Fig 4. (a) Postoperative transesophageal two-dimensional four-chamber view in a 12-year-old, 50-kg boy with tetralogy of Fallot and complete atrioventricular canal. The two patches used to repair the septal defects are evident (arrows). *(b) The same image with color Doppler shows a regurgitant jet (mosaic systolic pattern) into the left atrium due to residual mitral incompetence.* (LA = *left atrium;* LV = *left ventricle;* RA = *right atrium;* RV = *right ventricle.)*

temporary suspension of monitoring during cardiopulmonary bypass. Whether the probe should be withdrawn in children weighing less than 20 kg or during deep hypothermic circulatory arrest is still not clear; however, we believe the latter may be a useful precaution despite the safety of prolonged use of TEE as demonstrated experimentally by the absence of injury to the esophageal mucosa even in small animals [11].

The results of this study, although performed in a group of patients with a wide spectrum of cardiac anomalies, show the potential advantages of the intraoperative use of TEE. Transesophageal echocardiography is probably not needed when dealing with simple congenital heart defects such as an atrial septal defect; nevertheless, application of this technique in these patients has helped to achieve enough expertise, especially from the technical point of view, enabling us to confidently employ it in more complex cases and in progressively smaller children. The benefits of TEE, greatly enhanced by the addition of color-flow mapping, are particularly evident for patients with complex intracardiac anatomy who require elaborate surgical procedures. Intracardiac flow abnormalities, for example, can be easily detected, thus rendering TEE equivalent to an intraoperative angiography. This appears particularly helpful in detecting the presence and exact site of any residual intracardiac shunt and the amount of any transvalvular reflux, thus allowing the surgeon to make the most appropriate decision for each individual patient before discontinuation of cardiopulmonary bypass. This occurred in 1 of our patients in whom partial detachment of the atrial partitioning patch was detected after a modified Fontan procedure.

Transesophageal echocardiography permits uninterrupted monitoring of cardiac performance throughout the surgical procedure and therefore evaluation of the effectiveness of any inotropic or vasoactive drug administered [12]. This clearly represents an advantage over intraoperative epicardial 2-D echocardiography as poor surface contact and heart compression with consequent arrhythmias are avoided and temporary discontinuation of the operation is not required.

Nevertheless, TEE has some deficiencies that at the present time may be represented mainly by the limited number of scanning planes, which does not allow in most cases a clear visualization of structures such as the right ventricular outflow tract and the pulmonary valve. Furthermore, despite the fact that in all our patients TEE monitoring was judged to be satisfactory, in some cases, particularly when the heart rate rose above 140 beats/min, color signals derived from fast-moving cardiac structures temporarily impaired the quality of images. The low signal to noise ratio observed in such situations may probably be solved with more sophisticated probes especially designed for small children, as recently reported by Kyo and colleagues [7].

Our preliminary data suggest that TEE is a valuable tool to be used intraoperatively in children during open heart operations, particularly during correction of complex congenital heart malformations. From this experience it appears that a standard, commercially available, adult probe can be safely used in children weighing more than 20 kg. However, the amount of information obtained by the surgeon with this technique should favor its routine use and stimulate development of probes to be employed even in small infants and newborns.

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Swan-Ganz Catheter-Induced Massive Hemoptysis and Pulmonary Artery False Aneurysm

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Swan-Ganz catheter-induced massive hemoptysis and later pulmonary artery false aneurysm occurred in a patient with prosthetic mitral regurgitation. This patient was successfully managed by double-lumen endotracheal intubation, control of pulmonary hypertension, reversal of anticoagulation, mitral valve re-replacement, and transcatheter embolization. The pertinent literature is reviewed.

(Ann Thorac Surg 1990;50:644–6)

Massive hemoptysis secondary to pulmonary artery perforation by a Swan-Ganz catheter has been increasingly reported [1–3]. False aneurysms often develop in those who have survived [4–7]. Unlike bleeding from systemic bronchial circulation, hemoptysis from pulmonary artery perforation and false aneurysm formation involves the relatively low-pressure pulmonary arterial system. What constitutes the optimal management of such patients remains to be defined. This report presents our experience in the successful management of such a patient in whom both massive hemoptysis and false aneurysm developed after catheter-induced pulmonary artery perforation. Guidelines for the management of catheter-induced pulmonary artery perforation are outlined, and the literature is reviewed.

A 75-year-old woman who had a mitral valve replacement 12 years ago using a porcine prosthesis was admitted in progressive congestive heart failure. A new cardiac murmur consistent with mitral regurgitation was audible. She underwent cardiac catheterization that showed 4+ mitral regurgitation, dilated left ventricle, and normal coronary arteries with pulmonary arterial pressure of 65/25 mm Hg. During the attempts to wedge the pulmonary artery, the massive hemoptysis suddenly developed and the patient coughed up several hundred milliliters of dark blood; she soon after became hypotensive and experienced respiratory distress. Immediate fluid resuscitation was carried out, the tip of the catheter was pulled back, and partial heparinization was reversed. She was immediately intubated with a left-sided double-lumen endotracheal tube through which adequate ventilation was achieved. Intravenous nitroglycerin and nitroprusside drip were initiated to reduce the pulmonary hypertension. Hemoptysis ceased. She was extubated and all intravenous medications were tapered over 48 hours. Her congestive heart failure was partially controlled with digoxin, furosemide, and captopril. Follow-up chest roentgenogram revealed new infiltration in the mid-right lung field.

Three weeks after hemoptysis, the patient was readmitted in congestive heart failure. The repeat chest roentgenogram now showed a 4 × 4-cm round mass centered around the right middle lobe with extension across the major fissure (Fig 1). A diagnosis of false aneurysm was entertained. At repeat mitral valve operation, a pulsatile mass was palpable at the medial aspect of the right interlobar fissure. Postoperatively, her pulmonary arterial pressure came down to 30/10 mm Hg and the patient enjoyed a normal convalescence. Two weeks after her repeat cardiac operation, the false aneurysm was successfully occluded by transcatheter balloon embolization [5–7]. One year later she remains well and her chest roentgenogram now shows complete resolution of the aneurysm (Fig 2).

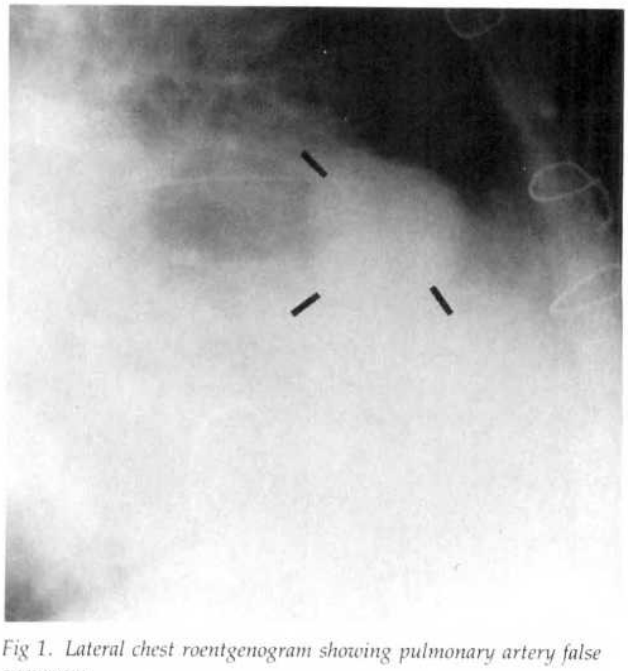

Fig 1. Lateral chest roentgenogram showing pulmonary artery false aneurysm.

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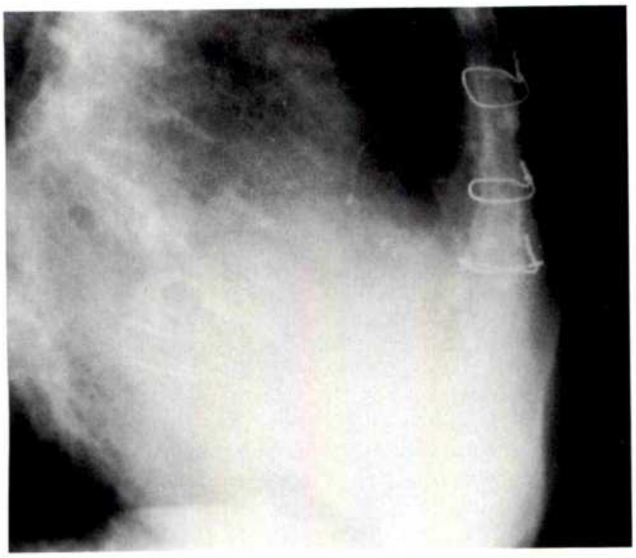

Fig 2. One year after balloon embolization, lateral chest roentgenogram showed resolution of aneurysm.

Comment

There are three major concerns in the management of patients with massive hemoptysis and later false aneurysm after catheter-induced pulmonary artery perforation: control of hemoptysis, follow-up measures, and treatment of aneurysm. Catheter-induced pulmonary artery perforation, fortunately, is relatively uncommon, with a reported incidence of 0.1% to 0.2% [3]. Unfortunately, when it occurs, it is associated with 50% mortality [1–3]. As most patients do not succumb secondary to exsanguination per se, but rather to aspiration and asphyxia, the initial management should focus on fluid resuscitation, airway protection, and arresting hemorrhage.

Endotracheal intubation using a double-lumen tube during the initial airway management has three advantages: protection of the contralateral lung, separate lung ventilation, and the ability to monitor bleeding. Unlike massive hemoptysis secondary to underlying lung disease, which originates from the high-pressure bronchial circulation, hemoptysis from catheter-induced pulmonary artery perforation may be controlled by modification of its risk factors. Three major risk factors have been identified: pulmonary hypertension, anticoagulation, and old age [1–7]. Essentially all patients had some degree of pulmonary hypertension, nearly two thirds received anticoagulation, and most were older than 70 years. Therefore, in addition to fluid resuscitation, double-lumen endotracheal intubation, reversal of anticoagulation, and lowering of pulmonary hypertension appear to be the reasonable initial steps in the managements of massive hemoptysis associated with pulmonary artery perforation. Almost invariably the bleeding tends to occur in the right lung. One should also consider placing the patient in the right lateral decubitus position.

The experiences using double-lumen endotracheal intubation for airway management in patients with this complication have been limited, but showed good results [8, 9]. Table 1 summarizes reported experiences in 5 patients including our own. All patients were old and had pulmonary hypertension. Double-lumen endotracheal intubation together with general supportive care and sedation led to spontaneous cessation of bleeding in all patients, and each one was able to be extubated within 48 hours. However, 2 patients bled again within a week; 1 died, and the other was managed successfully by transcatheter embolization. Based on the high risk of renewed bleeding, it seems advisable to perform a follow-up pulmonary arteriogram in patients who have recovered from massive hemoptysis to assess the degree of injuries as well as to outline therapeutic measures. Immediate thoracotomy and pulmonary resection after airway control probably are inadvisable in most instances because of frequent spontaneous cessation and patients' poor tolerance for emergent operation. However, thoracotomy and pulmonary resection should be reserved as a last resort when all measures fail to stop the bleeding.

In our particular patient, the management of her aneurysm was complicated by the urgent need for mitral valve replacement because of her congestive heart failure. Concomitant resection of the aneurysm was not carried out because of a substantial incremental operative risk. Because of the location of the aneurysm, resection would have required either a lobectomy or a major angioplastic procedure, which would have added considerable operative time and postoperative morbidity to an already difficult repeat mitral valve operation on a frail 75-year-old woman. Furthermore, her pulmonary hypertension would improve after the valve operation if related to acute

Table 1. Double-Lumen Intubation for Catheter-Induced Massive Hemoptysis: Patient Characteristics, Clinical Course, and Outcome

Patient No.	Age (y)	PAP (mm Hg)	Cessation of Hemoptysis	Extubation (h)	Outcome
1	90	35/16	+	48	Alive
2	89	45/15	+	36	Ventricular fibrillation; died
3	84	47/17	+	24	Rebled; died
4	83	34/13	+	30	Rebled; embolization; alive
5	75	60/25	+	48	Embolization; alive

PAP = pulmonary arterial pressure.

Table 2. Catheter-Induced Pulmonary Artery False Aneurysm: Clinical Course, Management, and Outcome

Patient No.	Events and Interventions	Outcome
1	Rebled in 7 months; embolization	Alive and well
2	Elective embolization 4 weeks later	Alive and well
3	MVR in 3 weeks; embolization 2 weeks later	Alive and well
4	Rebled; embolization	Alive and well
5	MVR in 1 week; rebled 3 weeks later; lobectomy	Alive and well

MVR = mitral valve replacement.

onset of heart failure secondary to degeneration of bioprosthetic valve. Such was the case.

Finally, one has to decide whether to treat the false aneurysm surgically or not. We believe that most catheter-induced pulmonary artery false aneurysms are amenable to transcatheter embolization because of their peripheral location. In fact, 4 of 5 patients reported [5–7] underwent successful treatment using transcatheter embolization technique for false pulmonary artery aneurysm (Table 2). One patient, however, underwent a lobectomy [4]. Despite a short-term follow-up, the transcatheter embolization technique demonstrated safety and effectiveness for treatment of catheter-induced pulmonary artery false aneurysm. Thus, it is the first choice of treatment whenever the anatomy is favorable.

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Esophagobronchial Fistula and Mediastinal Tuberculosis

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A 59-year-old man was seen with what preoperatively was thought to be an acquired esophagobronchial fistula secondary to an old burned-out infection with tuberculosis. At operation the gross and microscopic findings were most compatible with a congenital H-shaped esophagobronchial fistula. However, cultures of calcified lymph nodes grew *Mycobacterium tuberculosis*. The need for culturing calcified tissue to assure proper treatment is emphasized.

(Ann Thorac Surg 1990;50:647–9)

The association of acquired esophagobronchial fistula (EBF) in an adult with calcified mediastinal lymph nodes is generally presumed to be a chronic sequela of an old infection with tuberculosis or histoplasmosis [1, 2]. Aside from surgical repair, it does not require specific medical treatment. We report an unusual presentation of what appeared to be a congenital H-shaped EBF. Although the gross and microscopic findings were compatible with this, cultures grew *Mycobacterium tuberculosis*. The need for culturing calcified tissue specimens in apparently burned-out cases is emphasized to assure appropriate treatment. If not diagnosed and treated properly, infection may persist and lead to irreversible sequelae such as bronchiectasis.

A 59-year-old previously healthy white man was seen with a history of 5 or 6 episodes of syncope associated with cough during the preceding 6 months. Each episode was precipitated by drinking carbonated liquids. He was otherwise well and denied symptoms of chronic cough, fever, anorexia, weight loss, hemoptysis, night sweats, dyspnea, or chest discomfort. Five years before his presentation, he described a 3-week history of cough that had subsided with antibiotic therapy. His past medical, travel, family, and occupational histories and physical examination were unremarkable. A widened mediastinum and peribronchial thickening in the right middle and lower lobes was seen on his chest roentgenogram. A computed tomographic scan of the chest demonstrated enlarged and some calcified mediastinal lymph nodes and marked soft-tissue thickening adjacent to the esophagus below the level of the main carina. Skin tests to purified protein derivative (5 TU) and histoplasmosis were negative with positive controls. Three samples of sputum were negative for acid-fast bacilli by stain and culture. An esophagram demonstrated a right mid-EBF with aspiration of contrast material into the right lower lobe (Fig 1A). Esophagoscopy demonstrated a smooth depression that was lined and surrounded by normal-appearing squamous mucosa in the anterior esophageal wall at 28 to 29 cm; bubbles were seen refluxing in and out of this depression. Bronchoscopy revealed minimal extrinsic compression of the right main bronchus and a raised (2 to 3 mm), glistening, soft, white lesion on the posteromedial surface of the bronchus intermedius just distal to the right upper lobe take-off. The rest of the airways were normal.

At thoracotomy, a well-defined, smooth, benign-appearing H-shaped fistulous tract was found between the right proximal bronchus intermedius and the esophagus (Fig 1B). The mediastinum contained several large, hard, calcified lymph nodes; however, the fistula tract was not adherent to the nodes and was easily excised and repaired. A postoperative esophagram was normal. Microscopic examination of three lymph nodes that were removed revealed hyalinized nodules with no granulomata or inflammation. Microscopic examination of the EBF revealed a normal epithelium and very mild inflammatory changes consistent with a benign EBF. Special stains for acid-fast bacilli and fungi failed to detect these organisms in all the specimens. However, cultures of the calcified lymph gland grew greater than 500 colonies of *M tuberculosis*, sensitive to all antituberculous chemotherapeutic agents. The patient was treated with rifampin and isoniazid daily for 9 months and is asymptomatic 24 months after operation.

Comment

The large majority of acquired EBFs occur as a late complication of an underlying malignant process, most often carcinoma of the esophagus [3]. In a small number of patients, acquired EBFs are due to benign processes, but the diagnosis is difficult. The variety of congenital EBFs in adults has been emphasized in the literature [4]. Although EBF is a well-described manifestation of tuberculosis, some features of the case reported herein were unusual. The presentation with cough syncope following

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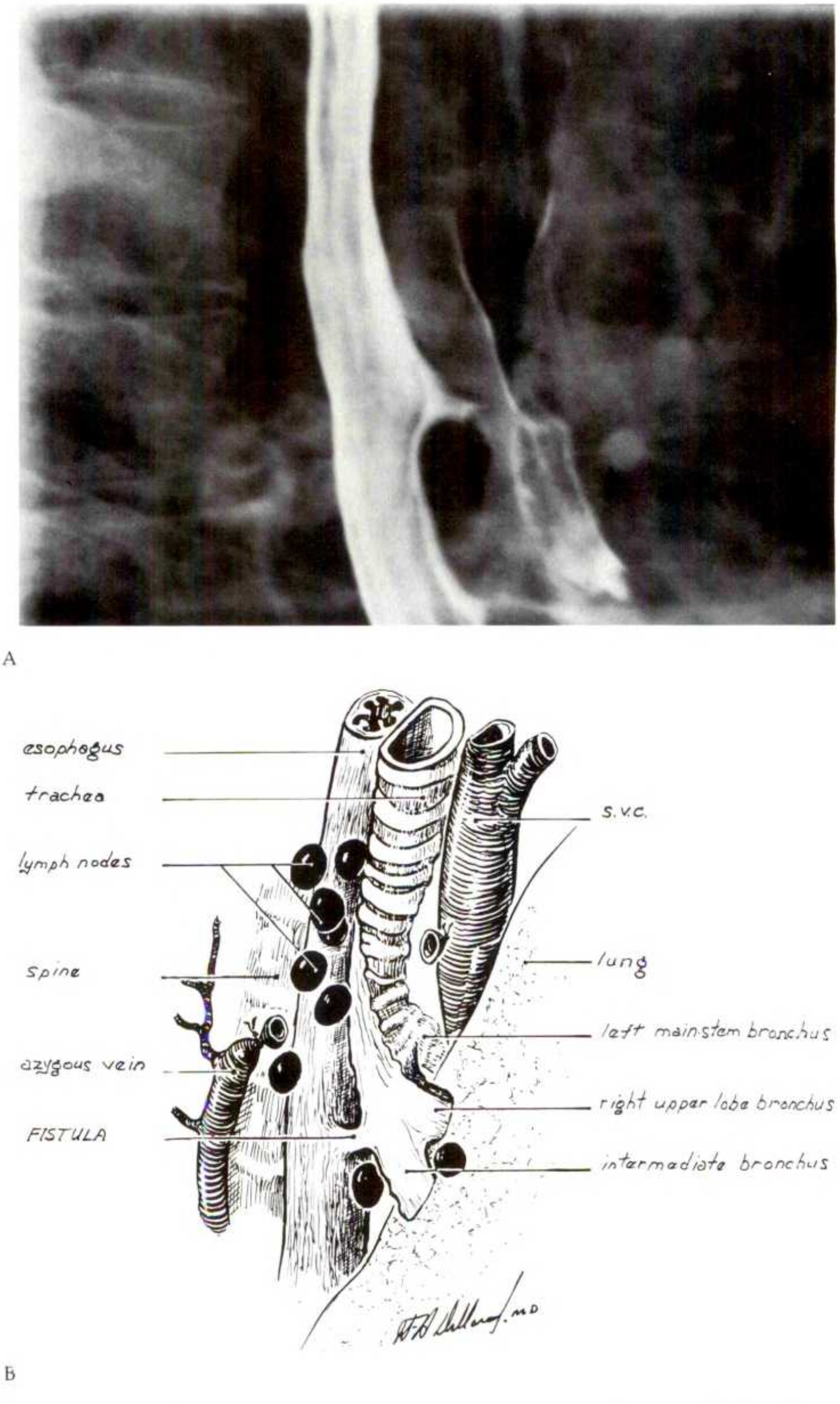

*Fig 1. (A) Esophagram demonstrating the fistula between mid-esophagus and right bronchus. (B) Findings at thoracotomy. Note the well-defined, benign-appearing, **H**-shaped fistulous tract between right bronchus intermedius and the esophagus.* (s.v.c. = *superior vena cava.)*

swallowing of liquids in an otherwise asymptomatic patient was consistent with a congenital EBF, and the gross appearance of the EBF was compatible with this diagnosis. The growth of *M tuberculosis* from a calcified lymph node lacking the microscopic features of granuloma or infection in a previously very healthy white male patient was unexpected.

In all of the previously reported cases of EBFs due to tuberculosis the diagnosis had been presumed or confirmed because of a clinical suspicion of active or chronic tuberculosis, previous exposure to active disease, positive skin tests to purified protein derivative, the presence of tubercule bacilli in sputum by stain or culture, or microscopic findings of tuberculous inflammation [2, 5–7]. The association of tuberculosis with EBF has therefore been relatively straightforward in those cases. In the case herein reported we documented growth of typical *M tuberculosis* from a calcified tissue that lacked any microscopic evidence of inflammation, acid-fast bacilli, or granulomata from a previously healthy man whose clinical presentation did not suggest even a remote exposure to tuberculosis. We would have assumed the cause of EBF in this case to be either congenital or a remote sequel of burned-out granulomatous disease and discharged the patient without antituberculosis therapy had we not processed the calcified lymph node for culture of tubercule bacilli. The index of suspicion for an active infectious cause and the need for culturing calcified tissue specimens in similar cases is emphasized as apparently burned out, inactive infections may indeed be still active and warrant appropriate medical treatment.

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Coexisting Thymic Carcinoid Tumor and Thymoma

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Thymic carcinoid tumors are unusual neoplasms that are different from thymomas. We report a case of coexisting thymic carcinoid tumor and thymoma associated with myasthenia gravis. The clinicopathological findings are discussed with a review of the literature.

(Ann Thorac Surg 1990;50:650–2)

Thymomas are composed of neoplastic, thymic epithelial cells and are associated with a variable number of lymphocytes. They are occasionally associated with clinical syndromes such as myasthenia gravis. Thymic carcinoid tumors, on the other hand, are unusual neoplasms that are composed of cells containing numerous neurosecretory granules. They show different morphological, functional, and behavioral characteristics than those of thymomas.

We present the clinicopathological findings of a 60-year-old man with a thymic carcinoid tumor and a thymoma associated with myasthenia gravis.

A 60-year-old man was admitted to our hospital having been diagnosed with myasthenia gravis (moderate generalized type) associated with an anterior mediastinal mass. On admission, physical examinations were normal except for myasthenic symptoms. Serial hematological and chemistry profiles were normal. The level of acetylcholine receptor antibodies was 1.95 nmol/L (normal, less than 0.16 nmol/L).

Computed tomography revealed a large cystic mass in the right anterior mediastinum and a small homogenous solid mass in the left adjacent region (Fig 1).

A thymectomy was performed. In the right lobe of the thymus, a well-encapsulated cystic tumor measuring 6 × 5 × 4 cm, which contained a dark brown viscous fluid, was found. The wall of the cyst was irregularly thickened. In the left lobe of the thymus, a solid tumor measuring 2 × 2 × 2 cm was found (Fig 2).

Microscopically, the cystic tumor was found to be thymoma, consisting of polygonal epithelial cells and a large number of lymphocytes. In contrast, the solid tumor was composed of anastomosing cords or solid nests of uniform round cells. The islands of tumor cells were surrounded by a fine fibrovascular stroma. Lymphocytes were limited to small isolated foci (Fig 3). No argyrophilic cells were found in either tumor by Grimelius staining. Several lymphofollicles with germinal centers were present in the thymus. On electron microscopy, the neoplastic cells of the solid tumor had numerous neurosecretory granules in the cytoplasm (Fig 4). These pathological findings of the solid tumor were compatible with those of thymic carcinoid tumor.

The patient recovered and no chemotherapy or radiotherapy was administered. A year after operation, there was no recurrence of the tumor.

Comment

Thymomas are the most frequent tumors of the anterior mediastinum, and are defined as neoplasms originating from thymic epithelial cells associated with lymphocytes in various proportions [1].

Thymic carcinoid tumors have long been confused with thymomas. However, primary carcinoid tumors of the thymus were first described in detail by Rosai and Higa in 1972 [2]. Thymic carcinoid tumors are unusual neoplasms that show different morphological, functional, and behavioral characteristics than those of thymomas [3].

As described in the literature concerning morphological features, thymic carcinoid tumors are composed of anastomosing cords, ribbons, islands, and rosettelike formations. These are composed of uniform round cells sepa-

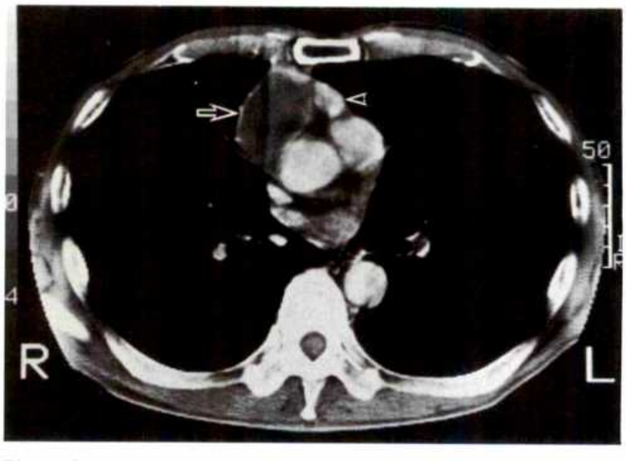

Fig 1. Contrast-enhanced computed tomography of the chest reveals a large cystic mass (arrow) *and a small solid mass* (arrowhead) *in the anterior mediastinum.*

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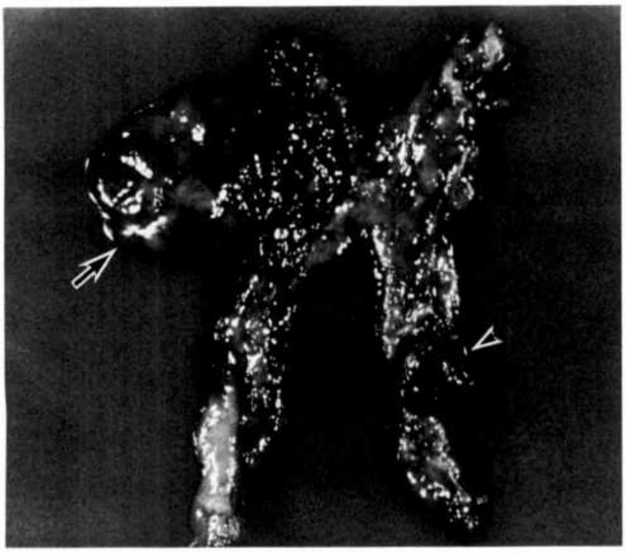

Fig 2. Resected specimen shows a cystic thymoma (arrow) *in the right lobe of the thymus and a carcinoid tumor* (arrowhead) *in the left lobe of the thymus.*

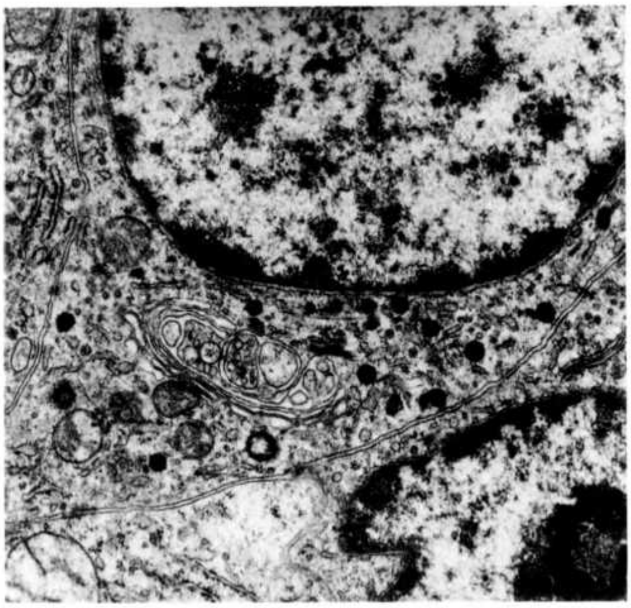

Fig 4. Electron micrograph: neoplastic cells of thymic carcinoid tumor are characterized by numerous dense-core, membrane-bound neurosecretory granules. (Uranyl acetate and lead citrate, ×15,000 before 36% reduction.)

rated by a fine fibrovascular stroma [1–6]. Distinction between thymic carcinoid tumors and thymomas may be complicated, particularly if the pattern is unusual. Electron microscopic observation is helpful in diagnosing thymic carcinoid tumors. The essential diagnostic ultrastructural feature in excluding thymomas is the presence of numerous neurosecretory granules [1, 3–6].

The clinical features of these tumors can also be recognized. Myasthenia gravis is the most common disease associated with thymomas [7], whereas endocrine abnormalities, including Cushing's syndrome, are frequently found in patients with thymic carcinoid tumors [3, 4, 6]. The presented case of the 60-year-old man was associated with myasthenia gravis. In contrast to the behavioral attributes of thymomas, thymic carcinoid tumors show metastases to regional lymph nodes or distant sites in a high percentage of cases [3, 4, 6].

The primary treatment of thymoma or thymic carcinoid tumor is operation. When the tumor is totally encapsulated, surgical excision is usually an effective cure [1, 3].

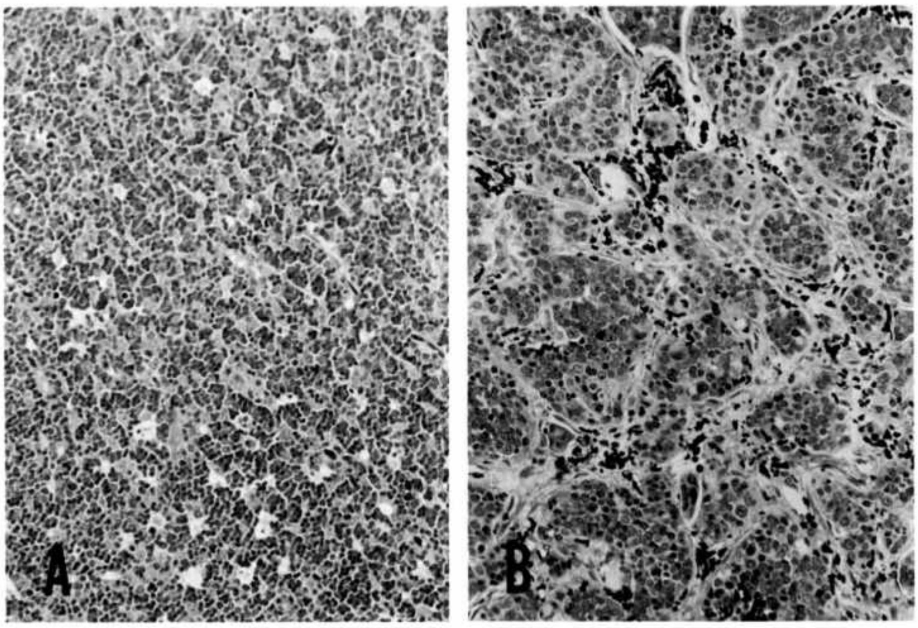

Fig 3. Microscopic photograph: thymoma (A) is composed of polygonal epithelial cells and large numbers of lymphocytes. Thymic carcinoid tumor (B) is composed of anastomosing cords or solid nests of uniform round cells. (Hematoxylin and eosin, ×50 before 34% reduction.)

In summary, we present a unique case of coexisting thymic carcinoid tumor and thymoma. Although the significance of coexisting thymic carcinoid tumor and thymoma remains unclear, the case indicates at least the possibility of coexistence.

We gratefully acknowledge Akemi Nagata for technical assistance.

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Cystic Medionecrosis of the Coronary Arteries and Fatal Coronary Vasospasm

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The histopathological basis of coronary vasospasm is not well defined. We report a patient with directly observed coronary artery spasm in whom cystic medionecrosis of the coronary arteries and great vessels and myxomatous degeneration of the mitral valve were evident. We suggest that myxoid connective tissue lesions of the heart may be linked to coronary vasospasm.

(Ann Thorac Surg 1990;50:653–5)

Coronary artery spasm has been recognized for decades. Although much clinical information has accrued, there is a dearth of pathological correlates.

We report a case of severe vasospasm of the right coronary artery (RCA), directly observed immediately after repair of a regurgitant myxomatous mitral valve (MV). Autopsy revealed lesions in the RCA and other coronary arteries similar to cystic medionecrosis of the aorta.

The patient was a 70-year-old woman with a long history of palpitations and with progressive dyspnea over the previous year. She recalled mention of a heart murmur and "mitral valve disease" approximately 25 years before admission. There was no family history of cardiac disease. Physical examination was unremarkable except for a grade 3/6 widely radiating, holosystolic murmur and an S_4 heart sound. Routine laboratory studies were within normal limits.

Cardiac catheterization revealed 4+ mitral regurgitation, normal coronary arteries, and normal left ventricular function. At operation, the patient was placed on cardiopulmonary bypass and hypothermic cardioplegic arrest was induced. The MV was exposed revealing a flail segment in the middle scallop of the posterior leaflet. A primary chorda of the anterior leaflet, directly opposite the flail segment, was elongated. Resection of the flail segment and repair of the MV was performed followed by insertion of a No. 28 Carpentier-Edwards MV annular prosthesis. The patient was weaned from cardiopulmonary bypass without difficulty, but palpation of the posterolateral wall of the left atrium revealed changes consistent with residual MV regurgitation.

Cardiopulmonary bypass was reinstituted and left atrial exploration revealed an additional elongated primary chorda of the anterior leaflet. Repairs were made and the atriotomy site was closed without incident. The patient was weaned from cardiopulmonary bypass. An intraoperative echocardiographic study demonstrated no residual MV regurgitation, but there was now evidence of a large hypokinetic region involving the interventricular septum and the inferior wall of the left ventricle. The location suggested a problem in the distribution of the RCA. An incision was made in the epicardium overlying the right main coronary artery proximal to the acute margin of the heart. The exposed RCA appeared taut, narrow, and stringlike, direct evidence of severe coronary vasospasm. Both vasodilators and calcium channel antagonists were administered, but the vasospasm persisted. Cardiopulmonary bypass was resumed and topical application of papaverine finally terminated the vasospasm. However, there was progressive impairment of ventricular function and systemic vascular collapse, which led to death in the operating room.

At autopsy, the majority of findings were limited to the heart, coronary arteries, and great vessels. External examination disclosed no marfanoid or acromegalic features. The coronary circulation was right dominant. The left main, left anterior descending, and left circumflex coronary arteries were grossly unremarkable, as was the majority of the dominant RCA, except for a small dissection noted within the proximal posterior descending artery. There was no luminal compromise by the dissection. No atheromatous lesions or thrombi were identified. The Carpentier-Edwards prosthetic ring was in place, and the MV repair was intact.

Histologically, pale blue to basophilic material, contained within variably sized cystlike spaces, was present disrupting the continuity of the media of several coronary arteries. These pools of acid mucopolysaccharide were most prominent in the posterior descending artery and RCA (Fig 1). In addition, similar focal cystic medial change was present in intramyocardial coronary vessels in all distributions. Cystic medionecrosis was evident in the ascending aorta and pulmonary artery. All valves showed myxomatous degeneration, particularly the mitral valve. Widespread circumferential myocardial contraction band necrosis consistent with acute myocardial reperfusion injury and infarction was evident in both ventricles (Fig 2). Focal, acute myocardial infarcts characterized by a neutrophilic infiltrate and coagulative necrosis of myocytes were present in the posterior left ventricle and interventricular septum. These infarcts were estimated to be less than 24 hours of age. Focal, remote myocardial

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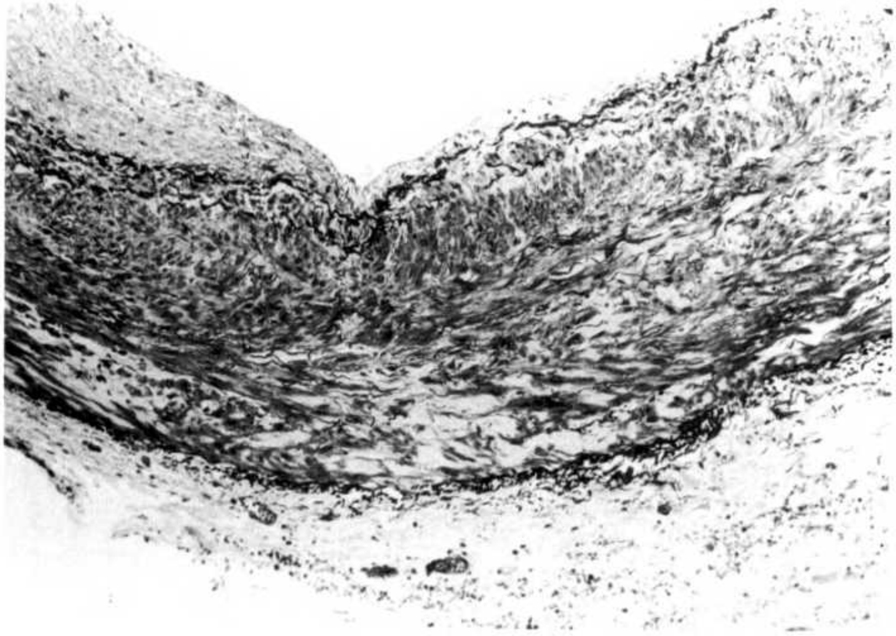

Fig 1. Low-magnification photomicrograph of the right main coronary artery demonstrating extensive disruption of media by multiple pools of mucin. Lumen is at top; adventitia is at bottom. Mucosubstance is the pale-staining material within the media. Elastic tissue appears black. (Movat pentachrome, ×20.)

infarcts were evident in the posterior left ventricle and right ventricle.

Comment

Cystic medionecrosis involving the coronary vessels is rare. Becker and Van Mantgem [1] identified a structural abnormality similar to cystic medionecrosis of the aorta in the coronary arteries of 4 patients known to have Marfan's syndrome. Cystic medionecrosis of the coronary arteries has been noted in 16 of the almost 60 case reports of isolated or primary dissection of the coronary arteries documented in the English-language literature, and as an incidental finding in 3 patients [2, 3]. The morphological correlates of coronary vasospasm are poorly defined.

Similar to the findings in our patient, an association between myxomatous (floppy) MVs, the pathological correlate of MV prolapse [4], and cystic medionecrosis of the aorta has been recognized; both lesions have been encountered in patients with Marfan's syndrome, individuals with acromegaly, and others without either of these disorders, suggesting an association between MV prolapse and systemic connective tissue abnormalities [5–7]. In addition, it has been shown that patients with MV prolapse have an increased incidence of coronary artery spasm [8]. The fatal intraoperative episode of vasospasm

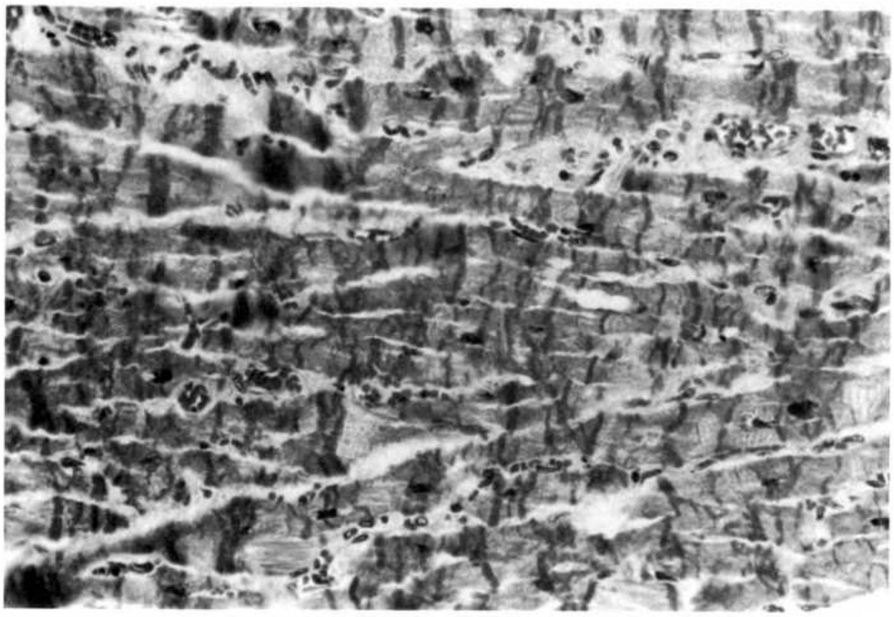

Fig 2. A section of myocardium showing a field of extensive contraction band necrosis. Contraction bands are the darkly staining lines of varying thickness oriented perpendicular to the long axes of the cardiac myocytes. (Hematoxylin and eosin, ×62.2.)

seen in our patient may have simply been a complication of the surgical procedure. However, the remote myocardial infarcts in the distribution of the RCA, in the absence of coronary atherosclerosis, are consistent with prior episodes of vasospasm. Our case report, along with the observations cited from the literature, suggests the possibility of a relationship between myxoid connective tissue lesions of the cardiovascular system and coronary vasospasm.

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Left Superior Vena Cava: A Pitfall in Computed Tomographic Diagnosis With Surgical Implications

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We report 2 cases in which computed tomography of the mediastinum demonstrated an abnormality originally misinterpreted as lymphadenopathy but subsequently shown to represent a left superior vena cava. Misinterpretation may result in errors in optimum treatment and may complicate surgical exploration of the mediastinum. These 2 cases are presented to remind radiologists and surgeons of the possibility of this unusual anatomy.

(Ann Thorac Surg 1990;50:656–7)

A 53-year-old man had a malignant melanoma completely excised from his left shoulder, after which he was routinely investigated for thoracic metastatic disease. Chest radiograph was unremarkable. A computed tomographic (CT) scan of his thorax, without intravenous contrast enhancement (Fig 1), performed 2 months later showed a left soft-tissue mass measuring 2.5 × 1.5 cm in the superior mediastinum, separated from the aortic arch and the pulmonary artery by a clear fat plane. This was considered to be consistent with an enlarged lymph gland, most likely representing metastatic malignant melanoma. In retrospect, however, the anomaly extended over two adjacent cuts (see Fig 1), suggesting its tubular shape. The patient was admitted for bronchoscopy and left anterior mediastinotomy. Results of bronchoscopy were unremarkable. Left anterior mediastinotomy was undertaken through the second intercostal space, disclosing a very deep chest with fatty infiltration of the anterior mediastinum. However, the aortic arch was clearly visualized, and the only structure in this position was a larger venous structure that was taken to be a left superior vena cava (SVC). Biopsy specimens were taken from normal-looking lymph glands, which proved histologically normal. The patient made an unremarkable postoperative recovery.

Our other patient, a 69-year-old man, was admitted for further investigation of lung cancer. He had a collapsed left upper lobe with an occluded left upper lobe bronchus seen by fiberoptic bronchoscopy. As part of our routine staging, a CT scan of his thorax with contrast medium (Fig 2) was performed, which showed a mass in the left superior mediastinum adjacent to the aortic arch. Left anterior mediastinotomy was undertaken and intense search for the left superior mediastinal mass was unsuccessful. Histology of the bronchial biopsy specimen confirmed the presence of squamous cell carcinoma. He proceeded to left thoracotomy and pneumonectomy, and was found to have a left SVC at operation. Surgical staging confirmed node-negative disease. His postoperative recovery was unremarkable.

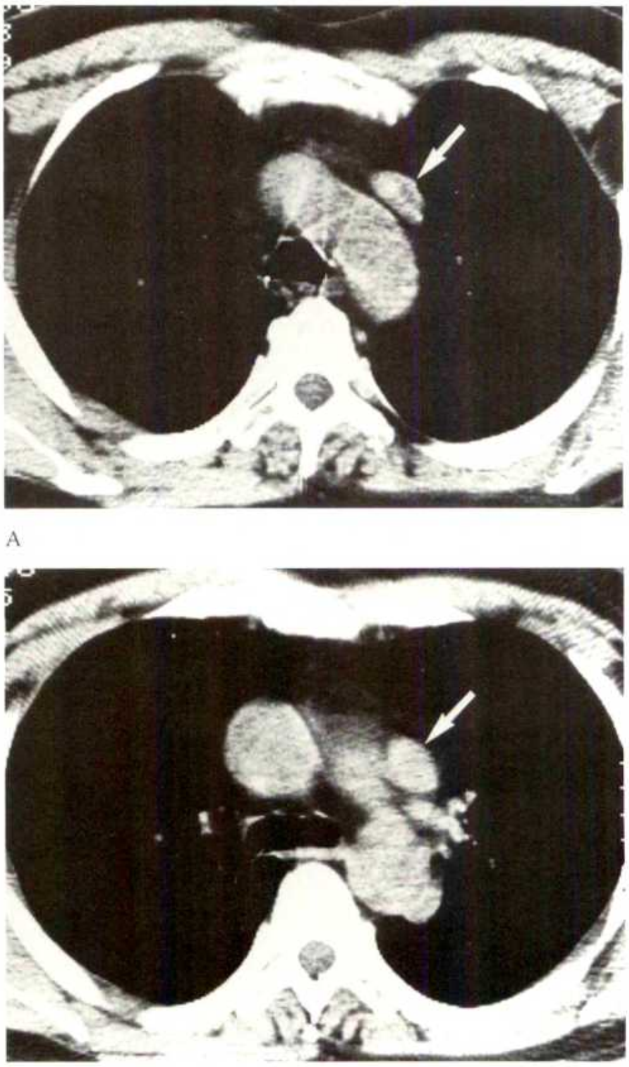

Fig 1. (A) Thoracic computed tomographic scan at the level of the aortic arch showing an oval opacity (arrow) *interpreted as an enlarged lymph node. It was found to be a left superior vena cava at mediastinotomy. (B) A lower cut showing the course of the left superior vena cava as it passes anterior to the pulmonary artery.*

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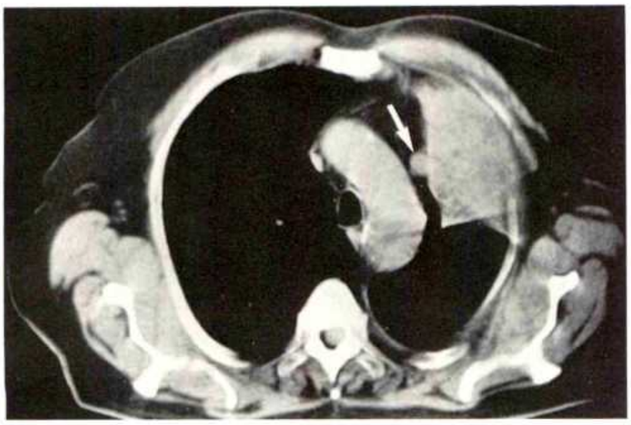

Fig 2. Computed tomographic scan showing a round opacity (arrow) *between the consolidated upper lobe and aortic arch. This was originally misinterpreted as an enlarged lymph node, but was shown to be a left superior vena cava at thoracotomy.*

Comment

The incidence of persistent left SVC varies from 0.3% in subjects with otherwise normal hearts to 4.3% in patients with congenital heart disease [1–3]. The embryology and anatomy of persistent left SVC have been well illustrated in the literature. The anterior and posterior cardinal veins unite into two short common cardinal veins (right and left duct of Cuvier) that empty into the sinus venosus of the heart. The persistent left SVC results from the failure of the left common cardinal vein to become obliterated. The left SVC descends vertically, anterior to the aortic arch along the left paramediastinum, and courses medially and drains into the coronary sinus. A right SVC may or may not be present.

Computed tomographic scanning is a very important noninvasive diagnostic tool in the staging and assessment of operability of thoracic neoplasms. Several studies comparing CT staging of the mediastinum with mediastinoscopy and postsurgical staging have shown sensitivities of 80% to 94% in detecting mediastinal adenopathy [4]. However, it is recognized that false-positive CT scans can occur [5], and CT evidence of lymphadenopathy is routinely assessed by cervical mediastinoscopy supplemented by left anterior mediastinotomy if the tumor lies in the left upper lobe or left main bronchus. During this evaluation enlarged nodes are biopsied. With the limited view available one could easily damage the left SVC if not forewarned of its presence and if instead one is looking for an enlarged node. With considerable experience with these techniques of mediastinal exploration we were fortunate in avoiding serious problems and improper assessment of the extent of disease.

We present these cases to raise awareness among cardiothoracic surgeons and radiologists of the appearance of vascular anomalies around the region of the superior mediastinum. The use of intravenous contrast medium to enhance vessels should be routine when performing CT scanning of the mediastinum.

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Liver Transplantation With Atrioatrial Anastomosis for Budd-Chiari Syndrome

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We report the case of a young woman with Budd-Chiari syndrome in whom mesentericocaval shunt was first performed, followed by transcaval liver resection and hepatoatrial anastomosis 3 years later. Liver transplantation became necessary 5 years later because of deteriorating liver function with portal hypertension and bleeding. Successful transplantation was performed with atrioatrial anastomosis with help of cardiopulmonary bypass, simplifying considerably the technical procedure and reducing dramatically blood loss.

(Ann Thorac Surg 1990;50:658–60)

Budd-Chiari syndrome (BCS) is a relatively uncommon group of disorders caused by occlusion of the major hepatic veins and/or the inferior vena cava (IVC), at or near the level of the hepatic vein ostia. Depending on the rapidity and the extent of the obstruction of hepatic vein outflow, the course of BCS may be rapid or chronic. Venoocclusive disease can mimic BCS but involves different vessels, mostly sublobular branches of the hepatic veins and hepatic venules. In recent years, effective surgical therapy has been developed [1–3] that is designed to decompress the obstructed hepatic vascular bed. As a result, the previously dismal outlook of patients with BCS has improved considerably. Portosystemic shunt offers best results in the chronic course of BCS with portal hypertension [2]. Liver transplantation in BCS remains controversial; however, the results are improving, and transplantation can be considered as a valid option in acute and chronic liver failure due to BCS or venoocclusive disease [4, 5].

We describe the case of a 37-year-old woman who was seen in 1978 with a large liver cell adenoma probably caused by contraceptives. Several months later BCS developed. No direct causal association could be evocated between presence of adenoma and subsequent development of BCS. Mesentericocaval shunt was performed in 1979 to decompress portal hypertension. The decision to perform a mesocaval shunt was based on anatomical situation during the operation and on the advantage of shunt construction away from the porta hepatis in this young patient, in whom further liver operation or transplantation could not be excluded at the time of the first operation.

The patient did well until 1982 when bleeding from esophageal varices appeared; radiological examination demonstrated occlusion of the shunt. In 1982 she underwent a dorsocranial liver resection with hepatoatrial anastomosis. In 1986 progressive liver failure developed and revision of the shunt was done with placement of an endoluminal prosthesis in the IVC to prevent compression through the hypertrophic caudate lobe. In 1987 recurrence of ascites appeared, with bleeding from esophageal varices and onset of encephalopathy. In June 1987 biopsy showed hepatic cirrhosis and the following liver function test values: prothrombin time, less than 20%, serum alkaline phosphatase, 250 IU (normal, 100 UI), serum glutamic-oxaloacetic transaminase 212 IU (normal, 67 IU), serum glutamic-pyruvic transaminase 268 IU (normal, 67 IU), and serum bilirubin, 43 μmol/L (normal, 7 μmol/L). Liver transplantation was decided on in June 1987 and the patient underwent orthotopic liver transplantation on August 17, 1987.

Inferior median sternotomy and median laparotomy were performed. Because of the previous cranial liver resection with hepatoatrial anastomosis, the decision to use cardiopulmonary bypass was made to facilitate the technical procedure and to reduce blood loss. Ascending aorta, superior vena cava, and infrarenal vena cava were cannulated and normothermic cardiopulmonary bypass was instituted. Recipient hepatectomy and resection of an inferior portion of the right atrium were performed, using a short period of electrically induced ventricular fibrillation for prevention of air embolism. The donor organ was prepared with a generous cuff of right atrium (heart transplantation with organ from the same donor was nevertheless possible). Standard anastomosis of IVC, hepatic artery, and ductus was performed with running sutures. Instead of infradiaphragmatic vena cava anastomosis, the diaphragm was split through the central fibrous body and the cuff of donor atrium was brought into the chest. Here a standard anastomosis to the defect in the right atrium, resulting from the removal of the previously anastomosed liver surface, was done (Fig 1).

The postoperative course was uneventful. An acute graft rejection on the 20th postoperative day was treated

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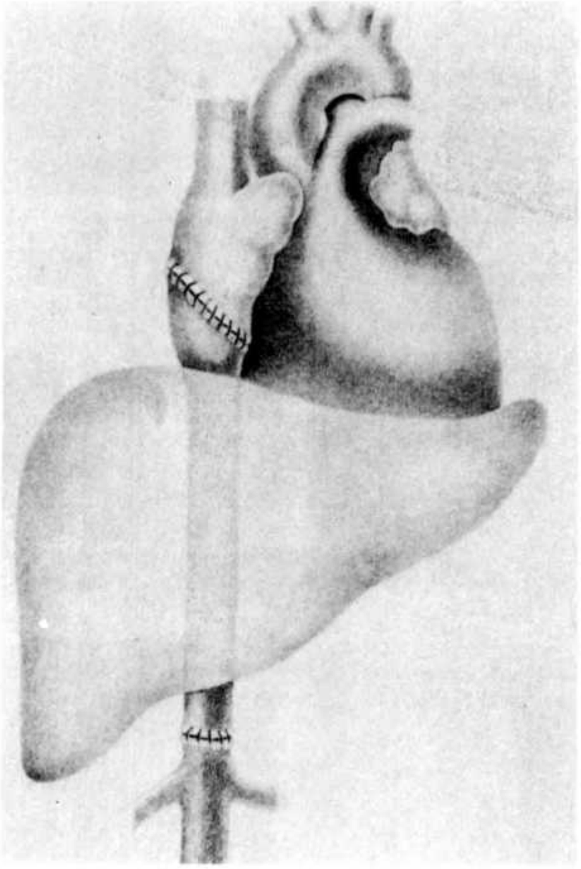

Fig 1. Orthotopic liver transplantation with proximal venous anastomosis in the chest.

with cyclosporine (according to plasma level), azathioprine (75 mg/d), methylprednisolone (1,000 mg/d), and OKT-3 (5 mg/d). The patient was discharged 6 weeks after transplantation. Two months later stenosis of the right hepatic duct was dilated percutaneously. Thirty months postoperatively the result in this young patient is very satisfactory: she is free of symptoms and liver function tests are normal.

Comment

Budd-Chiari syndrome is a syndrome of decreased hepatic outflow caused by varying degrees of hepatic venous occlusion. Hematological diseases that cause vascular thrombosis are the most common conditions that predispose to BCS. However, oral contraceptives, pregnancy, infections, connective tissue disorders, and malignant neoplasms are also involved in the pathogenesis of BCS. The increased intrahepatic pressure causes extravasation of plasma, dilatation of sinusoids, and congestion of the hepatic parenchyma. The end result is hepatic cirrhosis, similar to that observed in congestive heart failure. The reversibility of liver damage is a direct function of the rapidity and extent as well as the duration of hepatic venous obstruction. Early in the course of disease, relief of obstruction can be expected to result in reversal of parenchymal and hemodynamic abnormalities. The timing of therapy, therefore, has profound implications in regard to prognosis [1, 3].

Angiographic examination of the IVC and hepatic veins with pressure measurements can be combined with hepatic and mesenteric angiography; demonstration of occlusion of the major hepatic veins with or without involvement of the IVC and the typical spider-web pattern offers the best possibility of proper preoperative planning.

Common findings during operation are ascites, enlarged and congested liver, and splenomegaly. All patients have marked development of portosystemic collateral veins. In many authors' opinion, side-to-side portocaval shunt rather than transplantation is the treatment of choice in the acute and chronic stages of BCS, when liver function is still intact. Side-to-side portocaval anastomosis has been very effective in relieving BCS confined to the hepatic veins and in reversing liver damage. The majority of patients will recover and the shunt will remain patent permanently in most [2, 3]. Mesocaval and splenorenal shunts seem to be inferior to direct side-to-side portocaval shunt in the treatment of BCS [6]. The occlusion process in our patient extended into the great hepatic veins as well as into the adjacent IVC. This complication was treated successfully by dorsocranial liver resection with hepatoatrial anastomosis [7, 8].

Orthotopic liver transplantation seems to be indicated in patients with progressive hepatic failure often associated with a controllable predisposing factor [5]. There is little information available about the effect of a prior portosystemic shunt on liver transplantation. Portosystemic shunting is a traditional therapy for BCS but should be discouraged if the patient could be later a transplant candidate. Shunt dismantling is known to increase the technical complexity of the transplant operation. On the other hand, the distal splenorenal and mesocaval shunts have the advantage of construction away from the porta hepatis; this assumes importance if the patients become candidates for liver transplantation. Blood loss during liver transplantation will be lower than with any other type of shunt. Unfortunately, liver transplantation is technically more difficult when it is preceded by other operations performed in the right hypochondrium, especially after portocaval shunt. When it is combined with previous hepatoatrial anastomosis, a new similar anastomosis with the right donor atrium (because of the missing suprahepatic IVC) has to be performed, with additional technical difficulties. The most critical factor in dissection during recipient hepatectomy is blood loss.

Up to 1990, 110 patients with BCS have undergone liver transplantation. All patients had advanced and decompensated liver disease at the time of transplantation. Recurrence of thrombosis in the hepatic veins or the portal vein of the liver graft has been the major complication in addition to graft rejection. Long-term anticoagulant therapy has been advocated after transplantation in these patients [4].

Several techniques of circulatory support during liver grafting have been described. In our case, use of cardiopulmonary bypass enabled us to reinfuse the large amounts of blood lost during recipient hepatectomy, to facilitate the access to the right atrium for the proximal anastomosis, to reduce the dangers from the released potassium and acid radicals after first flushing of the liver, to integrate the function of splanchnicoaxillary venovenous bypass, to improve hemodynamic stability, and to reduce the time needed for operation.

Transplantation should be considered in patients with end-stage liver disease manifested by cirrhosis on biopsy and clinical symptoms and in case of sudden increase in extent of the thrombosis into the sublobular and centrolobular hepatic venules; in these cases drainage of the hepatic veins into the right atrium is not possible and only liver transplantation will be a definitive therapeutic option. Use of cardiopulmonary bypass facilitates greatly the surgical procedure in such complex cases.

Our report demonstrates that an aggressive therapeutic approach, requiring operations of escalating magnitude and multidisciplinary surgical treatment, can salvage patients who otherwise might not be considered candidates for operation. Further, it shows that patients who have had multiple previous shunt procedures should still be considered for liver transplantation.

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Resection of the Superior Vena Cava for Primary Lung Cancer: 5 Years' Survival

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We describe a patient with squamous cell carcinoma of the right lung that required a resection of the superior vena cava combined with a tracheal sleeve pneumonectomy. The superior vena cava was totally replaced with a polytetrafluoroethylene graft with 115 minutes cross-clamping of the superior vena cava. The patient remains healthy and the polytetrafluoroethylene graft remains patent 5 years 4 months after operation.

(Ann Thorac Surg 1990;50:661–2)

In this article we report on a case of squamous cell carcinoma of the lung that involved the superior vena cava (SVC) and trachea in a patient in whom replacement of the SVC with a polytetrafluoroethylene (PTFE) graft with simple cross-clamping of the SVC was performed along with a tracheal sleeve pneumonectomy. The patient remains well and the PTFE graft is still patent 64 months after the operation.

A 64-year-old man was examined in November 1984. He had been complaining of bloody sputum for about 2 months. A chest roentgenogram showed a mass shadow, 5 × 3 cm, in the right hilar area. A bronchoscopic examination revealed a solitary tumor in the anterior part of the trachea about 2 cm from the tracheal bifurcation concomitant with the main tumor extending up to the branching of the right main bronchus. Both lesions were pathologically diagnosed as squamous cell carcinoma. A computed tomographic examination revealed that the main tumor was causing a slight local compression against the posterior wall of the SVC (Fig 1).

On December 17, 1984, a right standard thoracotomy was performed. The tumor had grossly invaded the SVC, pericardium, phrenic nerve, and right main bronchus. However, there seemed to be no direct invasion of the trachea. The azygos vein was divided, and the relevant portion of the SVC, which measured 32 × 18 mm, was resected employing the temporary SVC–right atrial internal bypass technique [1]. The SVC defect was repaired with a pericardial patch. A right intrapericardial pneumonectomy was then performed. After this, the left main bronchus was divided right under the carina and the trachea was transected at the level of 3 cm (six rings) oral from the tracheal bifurcation, removing the segment of distal trachea that included the known solitary tumor. The airway was reconstructed by an end-to-end anastomosis of both stumps. After completion of the dissection of mediastinal lymph nodes, the chest was closed.

About 15 minutes after the patient adopted the supine position, a massive bleeding from the chest tube and rapid drop in blood pressure began. The chest was quickly reopened and pressure was applied to the middle mediastinum with a hand. This maneuver succeeded in stopping the active hemorrhaging and the patient was once again placed in a left lateral position. The pericardial patch was found to have detached from the SVC defect. Cross-clamps were placed proximally and distally to the SVC. Then, a total replacement of the SVC was performed using the PTFE graft, 10 mm in diameter and 7 cm in length. The SVC was cross-clamped for 115 minutes, during which the general condition of the patient stabilized except for a moderate swelling that appeared on the face and neck. The patient's return to consciousness was prompt and the postoperative course was uneventful. Anticoagulant therapy was not administered postoperatively.

The main tumor was located in the anterior segment of the right upper lobe and measured 5 × 3 × 5 cm. Microscopically, the tumor, composed of keratinizing squamous cell carcinoma, invaded the SVC adventitia. The solitary papillary tumor projecting into the distal tracheal lumen was also diagnosed as squamous cell carcinoma. This tumor, however, did not extend from the main tumor, and was situated in the mucosa. Therefore, the tumor was classified as multicentric carcinoma. A single tracheobronchial lymph node contained metastatic carcinoma.

After receiving a course of radiation therapy and chemotherapy, the patient was discharged 80 days after the operation. At present, 64 months after operation, the patient remains well with only a slight dyspnea after climbing stairs. A phlebographic examination in February 1990 showed patency of the reconstructed SVC (Fig 2).

Comment

Long-term survivors of malignant mediastinal tumors involving the SVC recently have begun to be reported after the introduction of new techniques for replacing the SVC with PTFE grafts with concomitant tumor resections [2, 3]. However, there have been few case reports of

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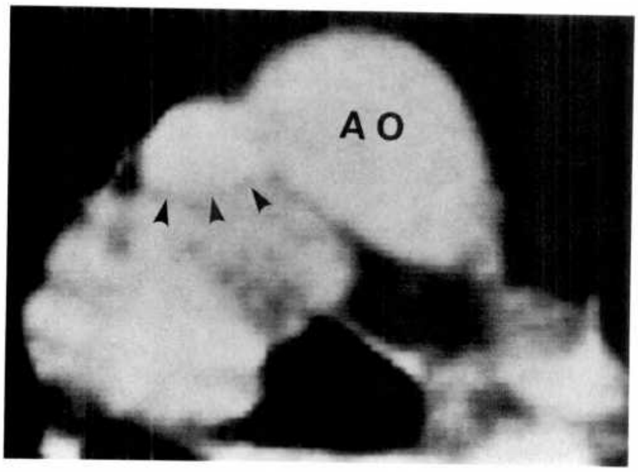

Fig 1. Superior vena cava is compressed from behind by the tumor as indicated by arrows. *(AO = aorta.)*

long-term survivors of primary lung cancer that invaded the SVC. In 1989, Nakahara and associates [4] briefly described the case of a patient with squamous cell carcinoma of the lung who underwent resection and replacement of the SVC with a PTFE graft combined with a completion sleeve pneumonectomy. Their patient was doing well 34 months postoperatively. In our case, the patient remains healthy 64 months after a similar operation. These reports encourage the aggressive resection of primary lung cancer involving the SVC in select patients.

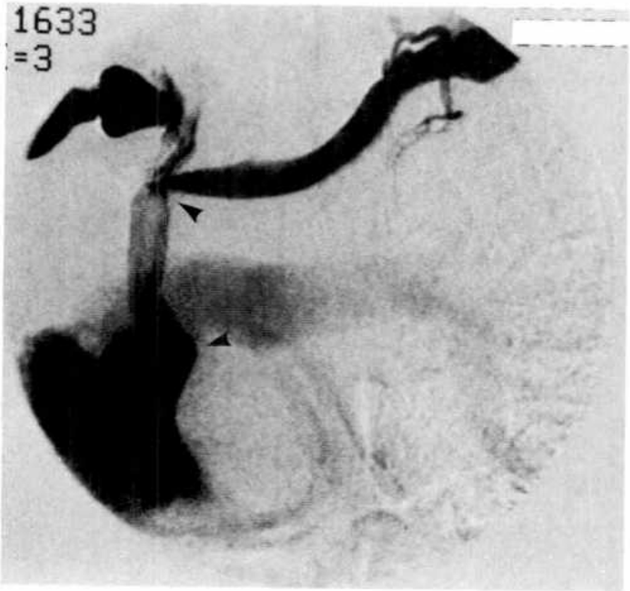

Fig 2. Arrows *show the patency of the polytetrafluoroethylene graft 5 years after operation.*

It is difficult in the present case to inquire into the original cause of bleeding from the repaired portion of the SVC because the pericardial patch had become detached from the defect of the SVC as a result of a postoperative mishap. The period of acute SVC obstruction with transected azygos vein lasted almost 2 hours and was tolerated quite well by our patient. A similar surgical misadventure was reported in 1954, in which the SVC occlusion time was about 1 hour but death occurred 30 hours later owing to respiratory insufficiency after a pneumonectomy [5]. When a patent SVC was electively replaced with a prosthetic graft, the SVC clamping time was reported to be 30 minutes [4], 45 minutes [2], 53 minutes [3], and less than 60 minutes [4], without remaining neurological deficiency. We could not find any case reports indicating neurological manifestations after a temporary SVC interruption. In an experimental study using cynomolgus monkeys, 1-hour clamping of the SVC with the ligated azygos vein proved to be safe [6]. The reconstruction of the SVC using a PTFE graft with the temporary cross-clamping of the SVC appears to be a safe surgical procedure.

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Polytetrafluoroethylene Graft for Spontaneous Coronary Dissection: 7-Year Follow-up

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Spontaneous coronary artery dissection remains an exceedingly rare cause of myocardial ischemia. The patients are usually young and female, and the dissection is frequently fatal. The use of polytetrafluoroethylene as an aortocoronary conduit is generally followed by early occlusion. We report a case of spontaneous right coronary dissection in which a polytetrafluoroethylene graft was placed that was observed to remain patent by angiography at least 72 months after operation.

(Ann Thorac Surg 1990;50:663–4)

Spontaneous coronary artery dissection is an unusual, typically fatal, cause of acute myocardial ischemia with only 85 reported cases [1] since Pretty's first description in 1931 [2]. Thayer and associates [1] found only 5 long-term survivors after operative intervention in their review. Only rarely has the absence of suitable autogenous tissue for aortocoronary bypass necessitated the use of various artificial conduits.

This report describes a case of spontaneous right coronary artery (RCA) dissection in which use of a polytetrafluoroethylene (PTFE) graft became necessary and in which the graft has remained patent by angiography 72 months after operation.

A 51-year-old previously healthy woman had severe chest pain followed by a cardiac arrest from which she was successfully resuscitated. Her history was remarkable only for an extensive bilateral stripping and ligation of both greater and lesser saphenous veins.

After resuscitation her heart rate was 72 beats/min and her blood pressure was 98/68 mm Hg. There were no stigmata of connective tissue disorders. Mild jugular venous distention was noted. The patient had a grade 1/6 systolic murmur and an S_4 gallop. Electrocardiography on admission revealed an acute inferolateral infarction. The peak level of creatine kinase was 4,240 U/L (0.07 MB fraction). Cardiac catheterization demonstrated a normal left coronary artery system, but a long mid-third filling defect of the dominant RCA was noted. Continued refractory intermittent myocardial ischemia resistant to maximal medical therapy necessitated urgent surgical intervention.

At operation an extensive search for suitable vein was fruitless. A rubbery firmness was palpable over the middle third of the RCA without obvious hematoma. The distal dominant RCA was believed to be too large to be adequately revascularized by the 2-mm right internal mammary artery. We elected, therefore, to use a 4-mm microporous PTFE graft (Impra; Impra Inc, Tempe, AZ). The RCA was obliquely transected and proximally ligated. An end-to-end distal anastomosis was created with 7-0 polypropylene suture followed by a proximal end-to-side anastomosis. The epicardium was closed over the distal graft to prevent kinking. Cardiopulmonary bypass was weaned without difficulty followed by a uneventful postoperative course.

Histology of the RCA segment (Fig 1) revealed dissection of an intact thickened intima, foci of cystic degeneration in the media, and subintimal polymorphonuclear leukocyte infiltrates. The patient was maintained on aspirin, 75 mg twice per day, and dipyridamole, 50 mg twice per day.

Six years after the initial procedure the patient had vague, atypical chest pain, and an equivocal stress test. A repeat coronary angiogram (Fig 2) was performed 72 months postoperatively and it documented a patent PTFE graft containing mild neointimal proliferation, but the anastomotic regions appeared free of excessive pannus formation. She is currently asymptomatic 7 years after operation.

Comment

The etiology of spontaneous coronary artery dissection remains obscure, but the female predominance and tendency to become manifest in the puerperium make a hormonal influence likely. The dissection is more common in the left coronary artery system, originating in the midportion of the media and extending between media and adventitia without intimal extension or flap [3]. Findings of cystic medial necrosis are present in about 30% of reported cases [1] with focal sarcolemmal ultrastructural changes noted at the dissection site by electron microscopy [3]. One current theory is that hemorrhage from weakened vasa vasorum into the outer wall of the media results in dissection [1].

The internal mammary artery followed by saphenous vein remain the preferred conduits for aortocoronary bypass, but in rare patients an alternative is needed. Cohn and Collins' [4] early use of PTFE coronary grafts resulted

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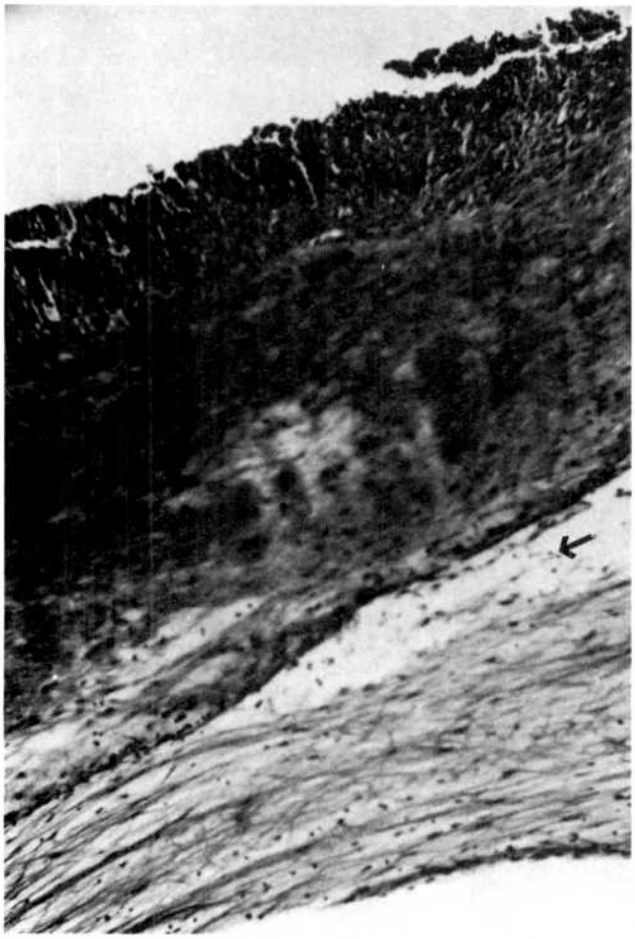

Fig 1. Longitudinal section of the right coronary artery. The dissection (arrow) *is through the media, which contains foci of cystic degeneration. (Hematoxylin and eosin, ×100 before 28% reduction.)*

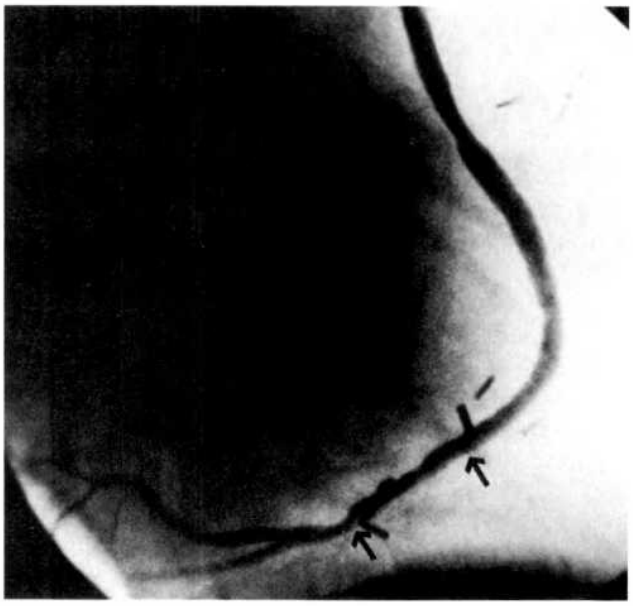

Fig 2. Angiogram obtained 6 years after operation showing patency of the polytetrafluoroethylene graft with mild neointimal proliferation sparing the anastomotic sites (arrows *indicate location of proximal and distal anastomosis).*

in 2 of 5 occlusions within 6 months, with the longest patent only 11 months [4]. They stressed the need for good runoff and high graft flow. Experimental work documented the need to use nonporous, "nonhealable" prosthetic graft material in the aortocoronary position to avoid the adverse effects of tissue ingrowth resulting from the unavoidable mediastinal hematoma [5]. In a series of 4-mm PTFE grafts with 30-μm diameter pore size and 8 weeks of coumarin anticoagulation after operation, patency was reported in two of three grafts studied at 24 months, but the one graft studied at 29 months had occluded [6]. Chard and associates [7] found that six of 28 PTFE anastomoses were patent after 3 years and noted that graft occlusion (observed at reoperation) was caused by bridging across side-to-side distal anastomoses by pseudointima. Experimental data show significantly increased patency of PTFE grafts in the aortocoronary position in animals given aspirin and dipyridamole postoperatively [8].

The long-term patency of the PTFE graft reported herein may be attributable in part to the relatively disease-free distal artery providing unimpeded runoff. The use of an end-to-end distal anastomosis may have also helped to avoid the pseudointimal bridging found by others to result in closure of distal side-to-side anastomoses [7]. Although PTFE is never preferable to autogenous tissue, we believe that this case supports the continued consideration of such a conduit when all other options have been exhausted, particularly if the runoff is good and graft flow can be expected to be high.

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Modified Pericardial Closure to Protect Cardiovascular Structures During Sternal Reentry

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A technique is described for covering the anterior surface of the heart and bypass grafts with autologous pericardium after myocardial revascularization. A trapezoidal flap is created that incorporates bilateral relaxing incisions to avoid distortion of grafts or increased risk of pericardial tamponade.

(Ann Thorac Surg 1990;50:665–6)

The increasing frequency of reoperative coronary artery bypass grafting has encouraged surgeons to seek methods of protecting vascular structures during sternal reentry. Different prosthetic materials and heterologous pericardial substitutes have been used to cover the heart, and different pericardial closure maneuvers have been proposed to prevent injury to the right ventricle, aorta, and patent venous and arterial grafts. The more frequent use of bilateral internal thoracic (mammary) arteries (ITAs) has stimulated questioning of the appropriateness of different conduits to bypass specific arteries. For example, in an effort to avoid crossing the midline with the ITA (which increases its vulnerability to injury during reoperation) some surgeons have felt compelled to use free ITA grafts instead of in-situ right ITA grafts to bypass the left anterior descending and circumflex system.

Closure of the pericardium over the anterior surface of the heart protects these structures, but pericardial approximation may lead to graft distortion and an increased risk of pericardial tamponade in the postoperative period.

In this brief report, a technique is described that protects the retrosternal cardiac structures and conduits without the potential disadvantages of direct pericardial closure.

Technique

The sternum is divided longitudinally in the usual manner, and the thymic fat is separated to expose the pericardium. The internal thoracic arteries are dissected from the chest wall; the pleural spaces are usually opened in this process. The pericardium itself is first incised horizontally just beneath the innominate vein and laterally toward both phrenic nerves (Fig 1A). The incision is then continued caudally along the course of both phrenic nerves medial enough to prevent nerve injury. A trapezoidal pericardial flap is thus formed, based on the diaphragm (Fig 1B). The pericardial flap is rotated and sutured tightly to wound towels over the abdomen to prevent shrinkage. Upon completion of the procedure, the pericardium is turned back over the heart and attached to the thymic fat with large hemoclips or sutures (Fig 1C). Retrosternal protection is thereby achieved, and two effective "relaxing incisions" allow entry of the ITA pedicles into the pericardial space without constricting the heart or impeding drainage. This technique has been used for 12 months on nearly 100 patients without complications. We have not yet had the opportunity to reoperate on any of these patients.

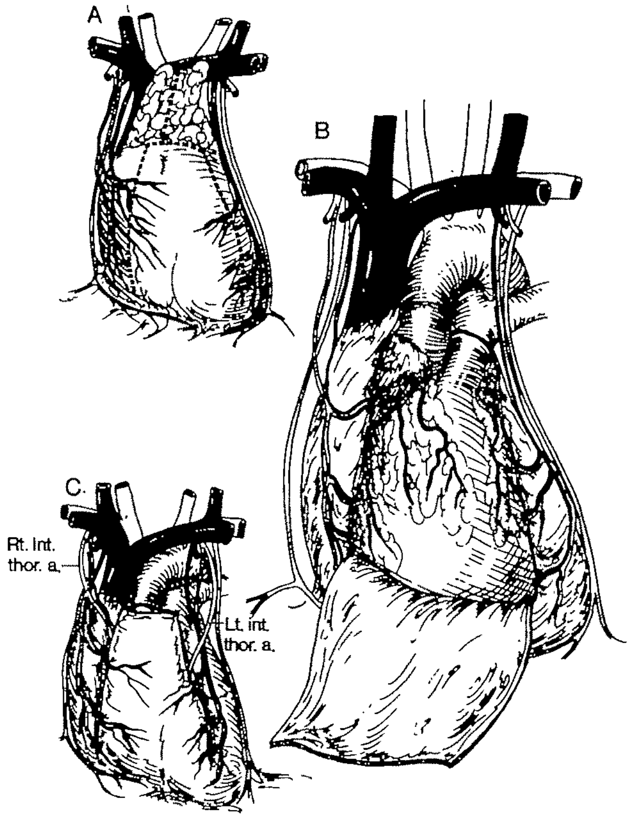

Fig 1. (A) Pericardial incision. (B) Pericardial flap. (C) Restored pericardium. (int. thor. a. = *internal thoracic artery.)*

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Comment

The value of covering the heart and conduits at the time of the first myocardial revascularization procedure has been a source of controversy during the past two decades. The problems of pericardial closure (eg, increased incidence of pericardial tamponade, kinking and compression of bypass conduits, and the adhesions formed around prosthetic or tissue pericardial substitutes) have hindered acceptance of these techniques.

The younger the patient, the greater the likelihood of another operation later in life, regardless of our attempts to provide them with atherosclerosis-resistant conduits. Although designed to protect all venous and arterial grafts, we believe that the technique described in this paper is especially applicable when conduits (eg, right ITA) cross the midline, because this decreases the probability of transecting the grafts at the time of reoperation.

Notice From the American Board of Thoracic Surgery

The American Board of Thoracic Surgery began its recertification process in 1984. Diplomates interested in participating in this examination should maintain a documented list of the operations they performed during the year prior to application for recertification. This practice review should consist of 1 year's consecutive major operative experiences. (If more than 100 cases occur in 1 year, only 100 need to be listed.) They should also keep a record of their attendance at approved postgraduate medical education activities for the 2 years prior to application. A minimum of 100 hours of approved CME activity is required.

In place of a cognitive examination, candidates for recertification will be required to complete both the general thoracic and cardiac portions of the SESATS IV syllabus (Self-Education/Self-Assessment in Thoracic Surgery). It is not necessary for candidates to purchase SESATS IV booklets prior to applying for recertification. SESATS IV booklets will be forwarded to candidates after their applications have been accepted.

Diplomates whose 10-year certificates will expire in 1993 may begin the recertification process in 1991. This new certificate will be dated 10 years from the time of expiration of the original certificate. Recertification is also open to any diplomate with an unlimited certificate and will in no way affect the validity of the original certificate.

The deadline for submission of applications is July 1, 1991. A recertification brochure outlining the rules and requirements for recertification in thoracic surgery is available upon request from the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Rapid Placement of the Hemopump and Hemofiltration Cannula

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Hypervolemia, a potential complication in patients on ventricular assist device support, can be managed by use of continuous arteriovenous hemofiltration. The Hemopump, a new catheter-mounted, transaortic axial-flow ventricular assist device, and the vascular access catheter for the Diafilter-30 Hemofilter system, used in continuous arteriovenous hemofiltration, are both usually inserted by way of the femoral artery. Because placing two large catheters in the femoral artery of a patient with peripheral vascular disease can compromise circulation, a technique for placing them in the abdominal aorta was developed. Two patients have undergone combined Hemopump and hemofiltration treatment, and neither experienced complications. Such techniques may benefit more patients in the future, as the usefulness of the Hemopump is proved.

(Ann Thorac Surg 1990;50:667–9)

Patients who are on ventricular assist device support may also require renal support to prevent or to treat hypervolemia. Continuous arteriovenous hemofiltration (CAVH) has proved useful in this setting [1] and offers advantages over acute hemodialysis because rapid shifts in intravascular volume are avoided. With the introduction of the Hemopump (Nimbus, Inc, Rancho Cordova, CA), a catheter-mounted, transaortic axial-flow ventricular assist device that is usually inserted by way of the femoral artery, a new CAVH technique was developed to avoid problems caused by placing two large-bore cannulas in the femoral artery of patients with peripheral artery disease.

Material and Methods

Reports describing the implantation techniques and clinical use of the Hemopump have been published [2, 3]. The CAVH system comprises a Diafilter-30 Hemofilter cartridge (Amicon, Danvers, MA) and a standard 11F dual-lumen catheter (Quinton Instrument Company, Seattle, WA) that serves as a venous return conduit. Percutaneous placement of a second 11-cm-long, 10F, single-lumen arterial cannula is also usually necessary to ensure adequate arterial inflow and filtration rates. The traditional system, therefore, generally requires several intravascular devices. In the technique described here, a single Quinton catheter is used for the arterial inflow conduit to avoid compromising lower extremity perfusion. Another Quinton catheter, or alternative cannula, is inserted in the contralateral femoral vein for venous return from the CAVH cartridge.

Hemopump Insertion Technique

The Hemopump is inserted by way of the retroperitoneal approach. An incision is made in the left lower quadrant to expose the distal abdominal aorta. After heparinization, the aorta is briefly occluded both proximally and distally so that a small aortotomy can be made. A 12-mm Dacron graft is then anastomosed to the aortotomy site with a continuous 4-0 monofilament suture, and the Hemopump and cannula assembly are passed through the graft. A silicone plug, which allows the Hemopump to be manipulated without causing bleeding, is placed at the end of the graft and secured with umbilical tape (Fig 1). With the use of fluoroscopic guidance, the cannula is advanced through the aorta and across the aortic valve, then positioned in the left ventricle. After the pump is in the proper position, pump flows are adjusted according to the needs of the patient.

Initiation of Continuous Arteriovenous Hemofiltration

The opening for the CAVH cannula is made by placing a 4-0 monofilament pursestring suture laterally at the distal end of the Dacron graft. A small stab incision is made in the center of the pursestring suture, and the 11F, 135-mm-long, dual-lumen Quinton catheter is advanced through the stab incision in the graft until its tip lies just inside the distal aorta (see Fig 1). The catheter is secured with the pursestring suture and then flushed. Pressure tubing, through which central arterial pressure monitoring can be accomplished, is connected to one of the ports, and the

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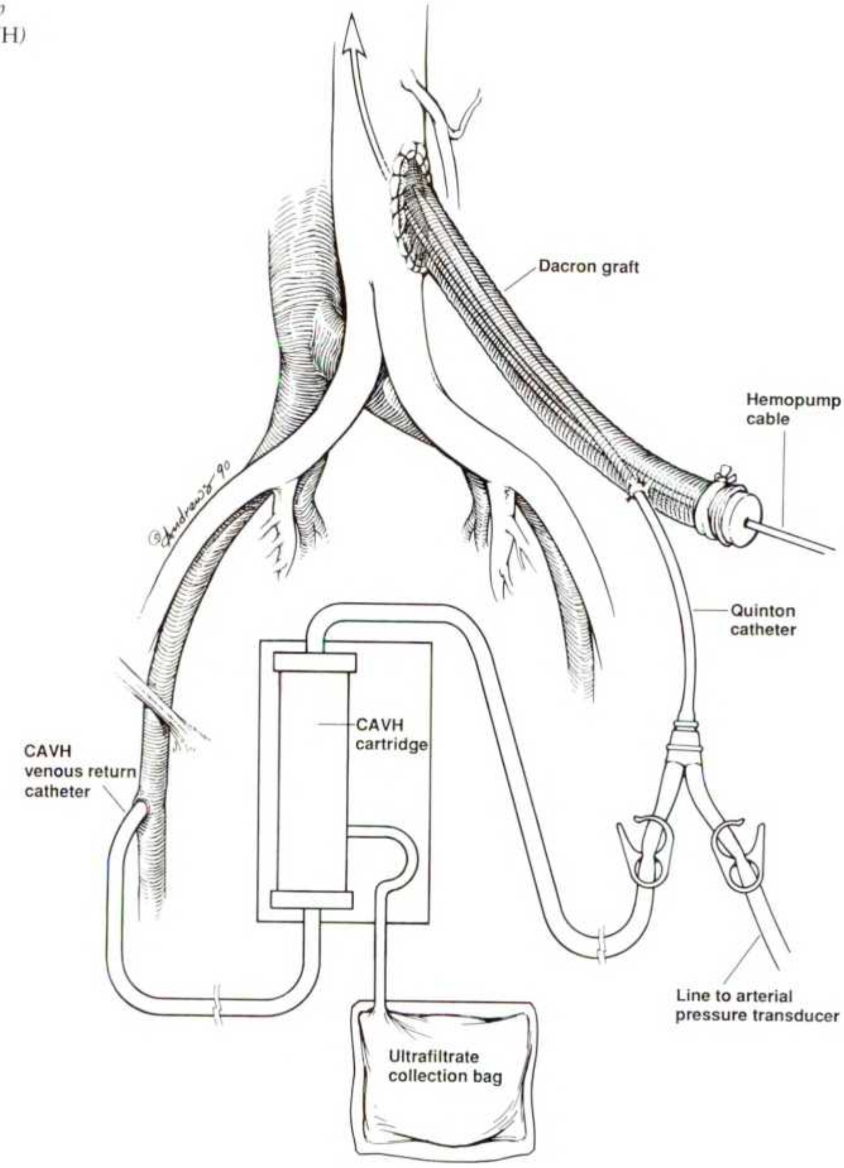
Fig 1. Retroperitoneal placement of the Hemopump and continuous arteriovenous hemofiltration (CAVH) system.


Dacron graft
Hemopump cable
Quinton catheter
CAVH cartridge
CAVH venous return catheter
Line to arterial pressure transducer
Ultrafiltrate collection bag
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CAVH tubing is connected to the other. Immediately thereafter, hemofiltration is begun.

After final adjustments to the position of the Hemopump are made, the plugged end of the graft is placed deep into the abdominal fascia. The incision is closed so that the Hemopump drive cable and the Quinton catheter exit from separate ends of the wound. A bulky dressing is used to prevent torsion of either device.

System Maintenance

Filtration rates (average, 250 mL/h) are controlled simply by raising or lowering the urinary collection bag in reference to the position of the filtration cartridge. If dialysis is necessary, dialysate fluid can be used in the contraflow direction through the cartridge. To prevent clotting, heparin is added to the cartridge so that partial thromboplastin time of blood within the cartridge is 70 to 80 seconds. The use of heparin in these quantities has only a mild effect on systemic anticoagulation. If heparin is contraindicated, citrate anticoagulation can be used. The optimal systemic perfusion pressure suitable for adequate performance of both devices ranges from 50 to 55 mm Hg.

Comment

Our technique for implementing CAVH in patients on the Hemopump allows rapid placement of both devices in the central arterial tree without further compromising lower limb perfusion. In addition to the use of a smaller hemofiltration cannula, which provides adequate flow to the cartridge, it is possible to monitor central arterial pressure. Hemofiltration rapidly corrects hypervolemia, which greatly simplifies postoperative management.

Both devices can be easily removed by withdrawing the catheters, excising the excess graft, and oversewing the remaining graft. If continued hemofiltration support is necessary, the Hemopump can be removed while the ultrafiltration catheter remains in place.

Combined treatment with the Hemopump and CAVH has proved successful in 2 of our patients. No complications, such as thromboembolism, infection, or bleeding, occurred.

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Notice From the Southern Thoracic Surgical Association

The Thirty-seventh Annual Meeting of the Southern Thoracic Surgical Association will be held at the Hyatt Regency Cerromar Beach, Puerto Rico, November 8–10, 1990. There will be a $155 registration fee for nonmember physicians except for guest speakers, authors and coauthors on the program, and residents. There will be a $50 registration fee for attendees of the Postgraduate Course, which will be held the morning of Thursday, November 8, 1990. The Postgraduate Course will provide in-depth coverage of thoracic surgical topics selected primarily as a means to enhance and broaden the knowledge of practicing thoracic and cardiac surgeons.

Advance registration forms, hotel reservation cards, and details regarding transportation arrangements will be mailed to Association members. Nonmembers should write to the Secretary-Treasurer, Gordon F. Murray, MD.

Gordon F. Murray, MD
Secretary-Treasurer
Southern Thoracic Surgical Association
111 East Wacker Drive
Chicago, IL 60601-

Improved Cannulation Method for Extracorporeal Membrane Oxygenation

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Extracorporeal membrane oxygenation has been shown to be useful for patients in reversible cardiogenic shock. Effective arterial cannulation techniques for infants have been developed that are simple to use and require minimal subsequent vascular repair or reconstruction after removal. Groin cannulation in adults frequently requires bidirectional arterial cannulation to ensure adequate distal perfusion as well as frequent complex arterial repairs after discontinuation. We describe a simple arterial cannulation technique using a single right-angle, high-flow arterial cannula. With this technique adequate bidirectional arterial perfusion is maintained with a single arterial cannula while the need for vascular repairs or reconstruction is minimized.

(Ann Thorac Surg 1990;50:670–1)

In acute-care medicine, situations occasionally arise in which patients are initially seen in and may persist in refractory cardiogenic shock or pulmonary failure. As an example, these may be patients with mitral valvar stenosis who, after mitral valve replacement, cannot be successfully weaned from cardiopulmonary bypass due to pulmonary hypertension and right heart failure. During this initial period, these patients may benefit from cardiopulmonary support while myocardial failure and pulmonary hypertension resolve.

The literature has argued for a combined approach of venoarterial extracorporeal membrane oxygenation (ECMO) coupled with pulmonary vasodilators (ie, prostaglandin E_1) to help treat this select group of patients [1–4]. Questions remain, however, as to the most ideal cannulation method to be used in placing adults on this bypass circuit. This report presents a simple method that has been found to be efficacious in achieving adequate circulatory support with anatomical preservation of the involved vessels.

Material and Methods

Initially, a standard groin dissection is carried out with isolation of the common femoral artery and vein. Meticulous homeostasis is mandatory.

The common femoral vein is cannulated first using a William Harvey 28 to 32F venous cannula, which can drain at a rate of 5.2 to 6.0 L/min. Initially, a 3-0 pursestring suture is placed in the anterior wall of the common femoral vein, approximately 2 cm long and 1 cm wide. This is secured with a Rumel clamp. The anterior wall of the vein is opened sharply within the pursestring and the cannula is introduced, advanced to the level of the distal inferior vena cava, and secured with the Rumel clamp. The venous cannula is then connected to the ECMO circuit (Fig 1).

Next, the common femoral artery is cannulated with either a Sarns (Sarns, Inc, Ann Arbor, MI) 5.2-mm or 6.5-mm right-angle, high-flow cannula, depending on the size of the common femoral artery. These cannulas allow delivery of 3.5 to 5.3 L/min of oxygenated blood. Similar to that in the femoral vein, a 3-0 rectangular pursestring suture is placed at the cannulation site and secured with a Rumel clamp. The femoral artery segment is isolated with clamps. The anterior wall within the pursestring is opened and the cannula is introduced, directed proximally, and then secured with the pursestring suture and Rumel clamp. The vascular clamps are then released. The cannula is then connected to the ECMO arterial circuit, and air is removed (see Fig 1). Both the arterial and venous cannulas are secured to the skin at multiple sites. The wound is closed after the instillation of 30 mL of fibrin glue. With proper selection of the arterial cannula sizes, distal extremity perfusion is maintained by arterial flow around the cannulas. Under these conditions, separate cannulation of the superficial femoral or profundus femoris arteries is unnecessary.

The ECMO circuit consists of a SciMed 4500 membrane oxygenator (Medtronic, Minneapolis, MN) with an integral heat exchange, a 1,300-mL SciMed venous reservoir, and a Bio-Medicus 540 portable pump console (Cardiovascular Resources, Inc, Broomfield, CO). The ECMO circuit is primed with 1,000 mL of Plasmalyte, 250 mL of packed red blood cells, 250 mL (12.5 g) of 5% albumin and 30 mmol of sodium bicarbonate. After the ECMO circuit is primed, the patient is connected to the circuit and ECMO is initiated. The ECMO flows are maintained between 2.5 and 7.0 L/min. A heparin drip is connected to the circuit to maintain an activated clotting time between 175 and 250 seconds.

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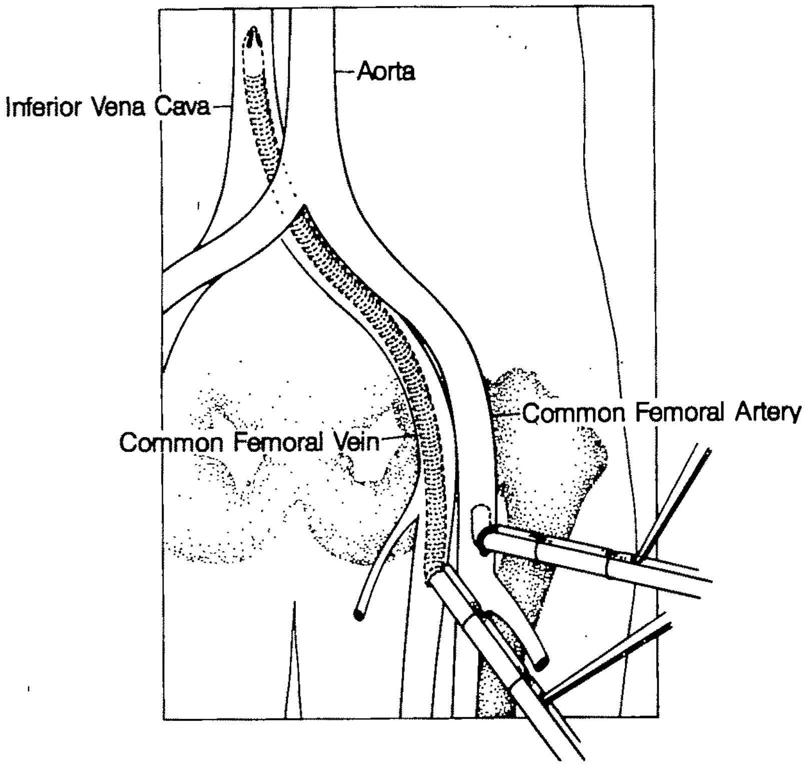

Fig 1. Modified femoral cannulation for extracorporeal membrane oxygenation. A high-flow, right-angle arterial cannula is placed in the common femoral artery. The cannula is slightly smaller than the internal diameter of the artery and is secured in position with a pursestring suture. This configuration allows for both adequate systemic and distal extremity perfusion while minimizing arterial trauma and the need for complicated repairs.

When cardiopulmonary support is no longer needed, the cannulas are clamped and removed. The pursestring suture in the femoral vein is simply tied, and a primary femoral arterial transverse repair is performed. Rarely are more complex vascular repairs necessary.

Comment

Prolonged cardiopulmonary support after operation on patients with reversible cardiogenic shock or pulmonary failure can be provided with extracorporeal membrane oxygenation. From the literature, it appears that the majority of recent successful experience is with neonates. However, this technique is becoming more frequently used with adult patients. Cannulation techniques for the neonate have been well described and appear to be both safe and efficacious [5, 6]. However, cannulation in the adult, via the groin, has proved to be somewhat more problematic and less satisfactory because of the need for distal extremity perfusion and subsequent complex arterial repairs after decannulation. We have developed a simple groin cannulation technique for the use of ECMO that provides adequate flow rates while obviating the need for distal extremity cannulation and detailed vascular repairs at the time of removal of the cannulas. We have found that the high-flow, metal-elbowed 5.2-mm or 6.5-mm arterial cannula, slightly smaller than the internal diameter of the common femoral artery, facilitates backflow, preventing distal extremity ischemia. Additionally, upon removal of the small-diameter arterial cannula, a simple primary vascular repair is all that is required. Currently, we have used this technique successfully in several patients for up to 5 days on ECMO without arterial erosions or other clinically significant complications. With this technique, the application of ECMO technology to patients with cardiopulmonary failure is facilitated while the concomitant morbidity is substantially reduced.

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Origins of Intraaortic Balloon Pumping

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In 1968, the *Journal of the American Medical Association* published a paper on the "Initial clinical experience with intraaortic balloon pumping in cardiogenic shock," which gave the results of the first clinical use of the procedure developed in our laboratory. For more than 2 years, our group was the only one using intraaortic balloon pumping clinically. Later, intraaortic balloon pumping was used by others, and despite poor clinical salvage, the hemodynamic benefit was demonstrated convincingly. At the present time, approximately 70,000 balloon pump procedures are performed annually.

(Ann Thorac Surg 1990;50:672–4)

Intraaortic balloon pumping (IABP) was a product of an endeavor begun more than three decades ago to explore first the concept and then the uses of diastolic augmentation. In 1951, I worked at Western Reserve University as a research fellow in the Department of Cardiovascular Physiology under the direction of Dr Carl Wiggers. My brother, Arthur, (then on the Cornell physics faculty and later at the AVCO Corporation) and I performed a series of experiments that demonstrated some of the principles of diastolic augmentation [1].

This eventually led to a cooperative effort between my research group at Maimonides Hospital in Brooklyn and the AVCO-Everett Research Laboratory in Massachusetts. We were trying to develop a permanent mechanical assist device that would provide diastolic augmentation for the relief of chronic left ventricular failure. In 1966, after several years of animal studies, we implanted such an avalvular pump into the aorta of 2 patients with chronic, terminal left ventricular failure. One patient survived only 24 hours; the second survived 11 days, then died of cerebral embolus [2]. However, in terms of hemodynamic support, the experience with this device was impressive. This experience also pointed to further problems that had to be overcome before a permanent left ventricular assist device could become a reality.

We believed it was reasonable, and probably easier, to achieve a temporary assist device based on the principle of diastolic augmentation. Arthur and I had some differences as to how best to approach development of such a device. He decided that he preferred to work with a group at the Massachusetts General Hospital. I would continue to work independently with my group of engineering and surgical research associates at Maimonides Hospital. This resulted then in two parallel, but entirely separate, efforts.

Moulopolous, Topaz, and Kolff [3] had reported on their early experiments with an intraaortic balloon pump in 1962. At about the same time, Clauss and his colleagues [4] reported on similar experiments. Both groups used a latex balloon (condom) on a small plastic catheter, driven with carbon dioxide. The use of a latex balloon ensured that the device would be occlusive in the aorta, and the use of carbon dioxide suggested that the shuttle gas was probably moved too slowly through the narrow catheter. At any rate, after their initial papers, neither group reported any further studies.

By early 1967, we had enough experience in the experimental animal to show clearly the advantages of a nondistensible polyurethane balloon. The use of helium as the shuttle gas gave us sufficient transit speed to assure appropriate timing. Our experimental results in dogs were published in the *Transactions of the American Society of Artificial Internal Organs* early in 1967 [5]. We believed that the evidence was strong enough to warrant a clinical trial in a small number of carefully selected patients who were in cardiogenic shock, which at the time was almost always fatal. We then secured approval from the Medical Research Committee of Maimonides Hospital for the use of IABP in a limited number of human subjects.

On June 29, 1967, we were called to examine a patient whose course is described in a 1968 article in the *Journal of the American Medical Association* [6]. (Follow-up data are given in reference [7].) The patient was a 45-year-old woman who was comatose, anuric, cold, and cyanotic; her blood pressure was unobtainable. The patient was clearly near death. We inserted the balloon pump and she was pumped for about 7 hours; her condition stabilized, and the pump was removed (Figs 1, 2). The patient recovered and was subsequently discharged from the hospital. Two other patients, however, did not fare that well. Both died; in 1 patient, we were unable to pass the balloon pump either from the femoral artery or from the axillary artery.

If our results with the first patient had not been successful, I feel fairly certain that we would not have had much support for attempting this procedure in other patients at Maimonides Hospital. I do not believe that would have meant the end of IABP, but it certainly could have delayed it for some years. The editors of the *Journal of the American Medical Association* have my gratitude and respect for accepting our paper for publication despite the meager clinical experience with just 3 patients.

By 1969, we received many inquiries from colleagues eager to try IABP. We thought that the only way to get a

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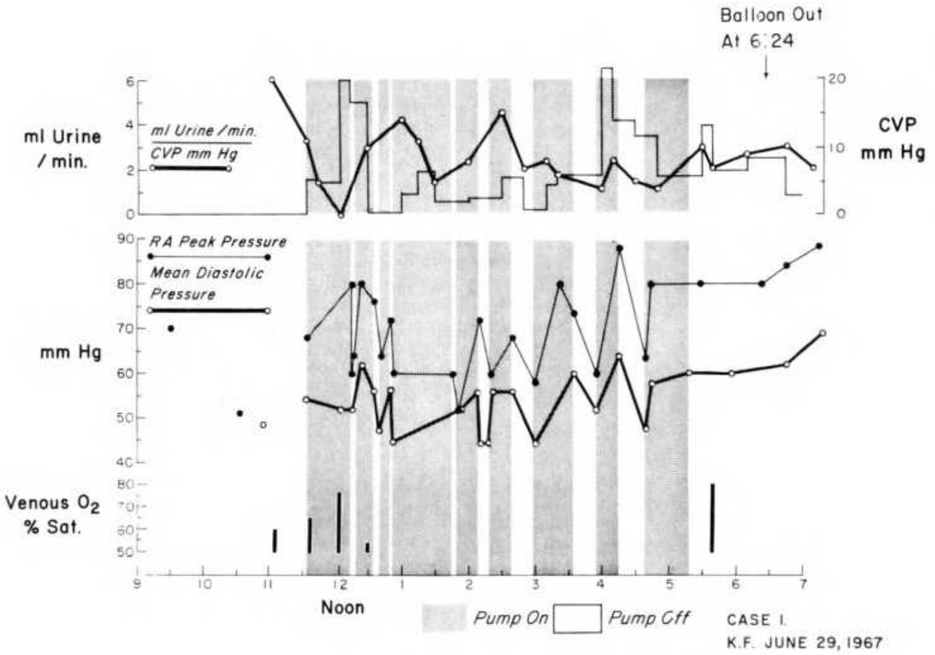

Fig 1. Graph of physiologic data recorded during pumping (case 1). Note increase in venous oxygen (O_2) percent saturation to normal levels after termination of pumping. (Low 12:30 PM *value probably resulted from technical error.) Pumping rapidly restored urinary output, which had been zero for several hours. (Reprinted by permission from* Journal of the American Medical Association *1968;203:135–40. © 1968, American Medical Association.)*

clear-cut answer on the clinical usefulness of this device was to organize a cooperative trial in which a considerable number of patients could be treated under a common protocol. Cardiologists and surgeons responded enthusiastically to my invitation to discuss a multicenter trial. They came from nine hospital and university medical centers: Albany, Barnes, Baylor, Peter Bent Brigham, Cedar-Sinai, Cornell, Duke, St. Vincent's (New York), and State University of New York (Downstate). The study participants prepared a common protocol and obtained approval from their respective institutional research committees. However, the members of the Maimonides Research Committee expressed doubt that the balloon pump really worked and vetoed the protocol.

Maimonides was a community hospital. Its resources were already stressed by being the site of the first heart transplantation in the United States and of the first implantation of a permanent left ventricular assist device,

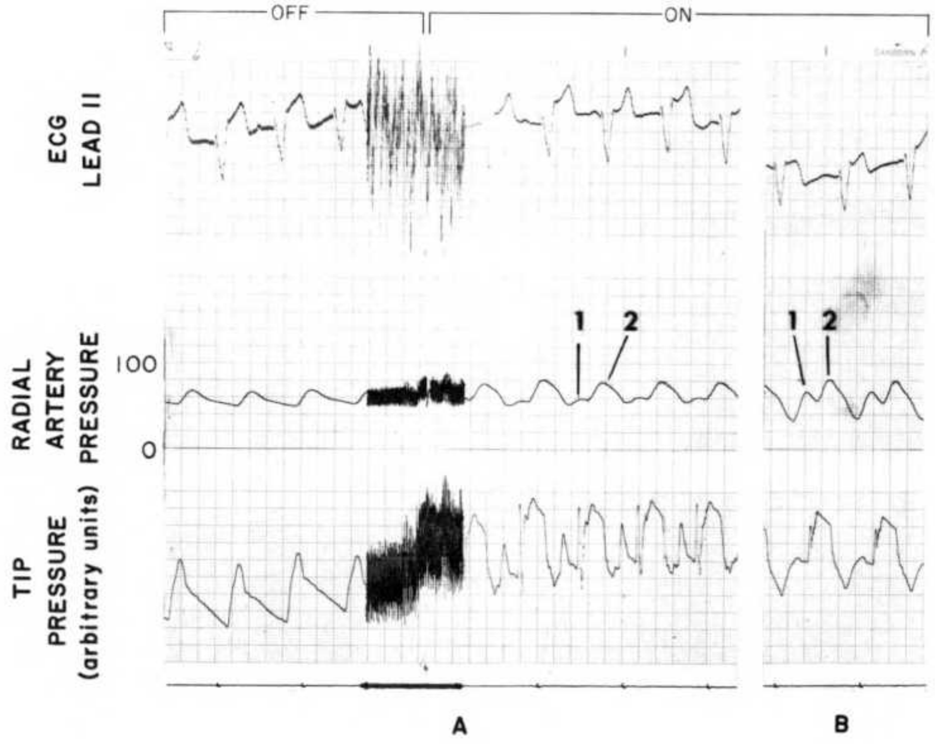

Fig 2. Electrocardiogram (ECG), right radial artery pressure, and central aortic pressure from pressure transducer (case 1). A, Recorded during initiation of balloon pumping. B, Recorded 15 minutes later. Component of radial artery pressure curve due to natural heart (1). Component due to balloon pumping (2). (Reprinted by permission from Journal of the American Medical Association *1968;203:135–40. © 1968, American Medical Association.)*

both by our group. A yet more difficult atmosphere in the hospital was created by the Research Committee action on the IABP protocol. The Board of Trustees, in their wisdom, encouraged me to look for a more hospitable environment.

In 1970, I left Brooklyn and came to Sinai Hospital of Detroit with our entire research group and a large portion of the surgical residency staff, 25 individuals in all. The John A. Hartford Foundation generously provided support for the cooperative study. Within a month, we started a clinical open heart surgery program and secured Research Committee approval to pursue, with the rest of the cooperating groups, further patient studies using IABP. Scheidt and co-workers [8] reported on the 87 patients treated in our cooperative study in the *New England Journal of Medicine* in 1973.

About 2½ years after our initial 1968 publication in the *Journal of the American Medical Association*, other groups began to report their early clinical experience. By that time, we had treated 30 patients with IABP [9]. At the April 1970 meeting of the American Society of Artificial Internal Organs, the group from the Albert Einstein College of Medicine described their use of a balloon pump in the treatment of 4 patients in cardiogenic shock. Only 1 patient survived [10]. In May 1970, the Massachusetts General Hospital-AVCO group reported their results in treating their first 8 patients in cardiogenic shock with IABP. They also had only one survivor [11]. So, after 2½ years of clinical experience with rather poor clinical results, it is not clear why the balloon pump effort just did not disappear. In retrospect, even though there was low salvage of patients in cardiogenic shock, the beneficial hemodynamic effects of IABP had been demonstrated convincingly and, therefore, it promised to be useful in other acute low-output left ventricular problems.

Any new idea, if it is to succeed, requires the courage and depth of understanding of its inventor to pursue the thought to its conclusion. The least challenges in the development of IABP were solving the many technical problems. More formidable were the intrainstitutional political agendas and their obstructive consequences. That IABP was not aborted in its infancy is witness to the remarkable support to us from the National Institutes of Health, from the John A. Hartford Foundation, from many residency and research staff members, and from independent-minded colleagues and investigators in the cooperative study, all of whom participated in this exciting adventure. Certainly in part because of their conviction, at the present time, some 23 years after the first clinical trials of IABP, there are approximately 70,000 such procedures performed annually.

A more complete description of these and related events appeared in the March 1987 issue of the *Transactions of the American Society of Artificial Internal Organs* as an article in the Moment in History series [12].

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CURRENT REVIEW

Guidelines for Transfusion Support in Patients Undergoing Coronary Artery Bypass Grafting

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We have reviewed the impact of evolving issues in coronary artery bypass grafting (CABG) on transfusion support for these patients. Issues include increased awareness of transfusion risks, reappraisal of traditional indicators triggering transfusion, and evolving alternatives to homologous blood transfusion such as autologous blood and pharmacologic therapy. These issues have been prompted by programs, such as the National Institutes of Health Consensus Conferences, to provide physicians with guidelines for appropriate use of blood components. However, evidence suggests that transfusion practice in coronary artery bypass grafting procedures remains variable and does not take into account the results of recently published clinical studies. We have therefore developed guidelines and recommendations for transfusion support in patients undergoing coronary artery bypass grafting. In summary, they are the following.

1. **Institutions with coronary artery bypass grafting programs should establish a multidisciplinary approach to use a combination of interventions designed to minimize homologous blood exposure.**
2. **Prophylactic transfusion of plasma and platelets are of no benefit and therefore carry an unnecessary risk to the patient.**
3. **Special request products such as designated blood donation from first-degree relatives should not be used because of the risk of transfusion-associated graft versus host disease.**
4. **For support of intravascular volume, crystalloids or colloids should be used because they do not have the potential to transmit infection.**

(Ann Thorac Surg 1990;50:675–83)

Open heart surgery is one of the most common major operations in the United States, with approximately 300,000 such procedures done annually. Because a large proportion of these procedures is accompanied by blood transfusion, they account for nearly 10% of the estimated 3.2 million annual recipients of red cell transfusions [1]. Although the use of coronary angioplasty for nonsurgical relief of obstruction has grown to 150,000 procedures annually, coronary artery bypass grafting (CABG) remains the most commonly performed cardiac procedure (230,000 per year) [2]. Approximately 800 US hospitals had open-heart surgical facilities in 1987, including over half of all hospitals with at least 400 beds [3].

Issues of blood safety in transfusion medicine, such as risk of retrovirus transmission in CABG [4], have renewed interest in the advantages of blood conservation [5–7], and have resulted in a reassessment of the benefits of routinely administered blood and blood components. These developments have been summarized recently by the National Institutes of Health (NIH) Consensus Conference on Perioperative Red Cell Transfusion [8]. Risks of blood transfusion, as estimated by conference participants, are shown in Table 1. Additional NIH consensus conferences have been held to address issue of transfusion practice for plasma [9] and platelets [10]. The general benefit-risk issues of blood transfusion therapy—ie, adequate tissue oxygenation and coagulation versus disease transmission and other adverse reactions—are of particular concern in cardiac surgery because of the medical risks of the patient population, the nature of the procedure, and the large number of blood components used [11]. Issues such as pharmacologic interventions that affect perisurgical blood needs [12–15], autologous blood predeposit as an alternative to homologous blood transfusion [16, 17], and changes in CABG surgical practice [18–20] have led to an evolution in blood transfusion practice in CABG. The purpose of this paper is to review the impact of these evolving issues and to generate blood transfusion practice guidelines for patients undergoing CABG.

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Table 1. Estimated Risks of Blood Transfusion

Factor	Risk
Human immunodeficiency virus	1:40,000–1,000,000
Hepatitis B	≤1:200–300
Non-A, non-B hepatitis	≤1:100
Fever, chills, urticaria	1:100
Hemolytic transfusion reactions	1:6,000
Fatal hemolytic transfusion reactions	1:100,000

From NIH Consensus Conference on Perioperative Red Blood Cell Use [8].

Hemostatic Evaluation of Patients Undergoing Coronary Artery Bypass Grafting

Preoperative hemostatic evaluation facilitates the diagnosis and treatment of bleeding disorders and decreases the likelihood of unsuspected hemorrhage during operation. Preoperative evaluation should include a history of previous transfusion/operation-associated bleeding, medication history including aspirin or other antiplatelet drug therapy, liver disease or known clinically significant alcohol intake, family history of bleeding problems, and patient history of easy bruising or unusual bleeding episodes. Laboratory testing should include a complete blood count as an estimate of red blood cell (RBC) mass to determine the adequacy of oxygen carrying capacity before operation, a platelet count to screen quantitative platelet adequacy, and a prothrombin time (PT) and partial thromboplastin time (PTT) to assess coagulation factor activity.

The value of a template bleeding time [21] preoperatively for predicting perioperative or postoperative bleeding is controversial. The potential value of the template bleeding time is to predict the risk of hemorrhage due to qualitative and also quantitative platelet disorders [21–23]. Although a prolonged template bleeding time is seen with a quantitative decrease in platelets [24], prolonged bleeding times in patients with platelet counts greater than 100 × 10^9/L (100,000/μL) are usually associated with platelet function defects [25]. The normal range for the bleeding time in most laboratories is 2 to 8 minutes. Clinically significant platelet deficiencies usually result in bleeding times in excess of 12 to 15 minutes [21, 22]. Many patients undergoing CABG can be predicted to have a platelet function defect acquired from aspirin therapy. A recent multiinstitutional survey revealed that 221/540 (41%) of patients scheduled for primary elective CABG were taking aspirin [26]. Second, patients undergoing CABG routinely acquire platelet function defects during cardiopulmonary bypass that persist for 1 to 2 hours afterwards [25]. Although most bleeding during open heart operations is related to platelet dysfunction rather than to deficiency of coagulation factors [25], acquired plasma coagulation defects may arise from the dilution by priming solutions for the extracorporeal circulation, transfusion of saline-washed cells, or multiple-unit transfusions [24]. As virtually all patients undergoing CABG are treated with heparin, the possibility of heparin-induced thrombocytopenia should also be considered in the thrombocytopenic patient [27]. The observation of a prolonged template bleeding time preoperatively in patients not treated with aspirin requires further evaluation to plan patient management. Factors to consider in addition to medications such as aspirin include clinical disorders such as von Willebrand's disease [28] or Glanzmann's thrombasthenia [29] that also affect template bleeding time. However, the potential usefulness of this test intraoperatively is limited in CABG because of the technical considerations of doing this test during operation with cardioplegia, where prolongation of the bleeding time has been demonstrated to occur in the setting of hypothermia.

Preoperative assessment of plasma coagulation by the PT and PTT is recommended to screen for any nonplatelet-related coagulopathy. The PTT, although recognized to vary markedly owing to different reagents and different lots of the same reagent, can, nevertheless, be useful in demonstrating defects in the (contact-activated) intrinsic pathway [30]. Similarly, the PT serves as a screening test in assessing the extrinsic portion of the coagulation system. Assessment of fibrinogen levels preoperatively is not done routinely, but may be helpful if complex operation, bleeding complications, or anticipated large-volume intraoperative cell salvage are anticipated. Although normal ranges for both hematologic and coagulation variables are wide, the knowledge of baseline preoperative levels can be useful in guiding subsequent treatment of unexpected bleeding [31].

Preoperative evaluation also includes pretransfusion compatibility testing and selection of appropriate units for transfusion. Sufficient time should be allowed for assessment of alloantibody and other serologic problems that could potentially complicate the cross-match, such as cold agglutinins. Routine hypothermia and cold cardioplegia is used in CABG, with the body maintained at 28° to 32°C and the myocardium at less than 15°C. Some centers favor blood cardioplegia with a solution having a hematocrit of 0.10 to 0.15. The presence of cold agglutinins should be considered [32, 33]. Although nearly all people have immunoglobulin M red cell autoantibodies reactive at 4°C, and some have antibodies that agglutinate RBCs in vitro at room temperature (18°C), cold agglutinins have virtually never been conclusively implicated as a cause of complications in these patients [34]. However, occasional individuals have strong cold agglutinins reacting up to 37°C, leading to concern about the possibility of systemic or intracoronary RBC agglutination during cardiopulmonary bypass in patients with unusually strong cold agglutinins. Cardiopulmonary bypass has also been modified in various ways for such patients—limited or no systemic hypothermia, warmed transfusions, avoidance of blood cardioplegia, and/or flushing out coronary RBCs with warm clear cardioplegia solution at the beginning and end of the procedure [32]—but the uncontrolled nature of these case reports leaves the benefit of these measures undefined.

Guidelines for Laboratory Testing

Screening tests to determine patients at risk for subsequent bleeding that would require therapeutic intervention should include complete blood cell count, platelet count, PT, and PTT. A positive patient history or physical examination should raise the consideration of a screening template bleeding time, fibrinogen determination, and/or transfusion medicine consultation for further diagnostic evaluation. All instances of suspected or documented platelet and coagulation factor abnormalities that are otherwise unexplained should be reconciled by further diagnostic procedures with the assistance of a transfusion medicine consultation. These can then be addressed either pharmacologically or by transfusion of the appropriate blood component. In elective CABG, ample time should be allowed for pretransfusion testing and for blood typing and cross-matching so that problems can similarly be resolved before operation.

No special low-temperature compatibility testing is necessary. However, it seems prudent that detection of an unusually strong cold agglutinin should lead to consideration of modifying the cardiopulmonary bypass procedure, particularly when blood cardioplegia or especially deep hypothermia is planned. If time permits, more detailed preoperative studies of the antibody's thermal amplitude, ideally using a blood sample kept at 37°C until serum separation, may help in planning the degree of systemic hypothermia used.

Approach to the Patient Undergoing Routine, Uncomplicated Coronary Artery Bypass Grafting

A recent multicenter transfusion practice audit study in patients undergoing routine, uncomplicated CABG showed that RBCs are transfused in 74% of all patients undergoing this procedure [26]. This same study reported transfusion rates of plasma and platelets that varied from 0% to 100% of patients at individual institutions, demonstrating significant variability in transfusion practice. Previous consensus conferences have demonstrated that prophylactic transfusion of plasma [9] or platelets [10] in this setting is not indicated; the focus of our review and guidelines in this section will therefore address issues related to use of RBC administration.

Red blood cell transfusion in CABG has historically been variable among institutions [11], and recent literature suggests that this variability still occurs [26]. In the early CABG experience, RBC transfusion practice was affected by issues such as the availability of blood [35], refinements in technical aspects of CABG [36], the development of intraoperative and postoperative autologous blood salvage and reinfusion [5, 37], and the acceptance of hematocrits between 0.25 and 0.30 related to hemodilution [11, 18, 38]. Clinical determinants of blood use during CABG have been well-defined by single-institution programs [18] to include age (>60 years), sex (female), initial circulating RBC volume (dependent on body surface area and hematocrit), reoperations, and more recently, double mammary artery implants [19]. Factors that some reports have shown to be unrelated to determinants of blood use during CABG include number of grafts [18], institution size, and the number of open heart procedures performed annually [11, 39]. However, variation in blood transfusion practice during CABG among institutions has been identified [11, 27, 40] that may be independent of previously identified patient-dependent determinants of blood use [18]. Surgeon specificity may affect transfusion needs through variations in amount of surgical blood loss or variations in the threshold for administering components. Single institutions have reported variances in the mean homologous blood transfusion from only 0.3 ± 1.4 units/patient (mean ± standard deviation) (44/441 [10%] of those undergoing consecutive primary CABG required transfusion) [18] in 1981 by one surgeon to a range of 8.0 to 2.0 units/patient from 1967 through 1984 for all surgeons at the same institution [41]. Similarly, the RBC transfusion needs of four different cardiac teams in the same city in 1987 varied from 4.4 to 6.6 units/patient, and were increased for each team when compared with 1982 [42]. Possible explanations for this variability could include surgeon specificity, but also include such factors as CABG after failed coronary artery angioplasty [43], reoperations [41], and changing patients' ages: the median age of patients who underwent CABG increased from 50 years in 1967 to 62 years in 1986, with 40% of surgical candidates aged greater than 65 years and 20% of surgical candidates aged greater than 70 years [20].

In addition to the above-mentioned determinants of RBC need, operative techniques have also had a major impact on blood transfusion practice in CABG [44]. The principal methods of conserving blood in patients undergoing CABG include using a nonhematic prime solution for the bypass pump [45], intraoperative autologous blood salvage and reinfusion from the operative field [46] and/or the extracorporeal circuit after cardiopulmonary bypass [47], postoperative collection and autotransfusion of chest drainage [48], and the use of nonhematic volume expanders and the acceptance of a normovolemic anemia [18, 49, 50]. A recent survey of 18 institutions indicated that 15/18 (83%) CABG programs used some form of intraoperative autologous blood salvage and reinfusion, and 5/18 (28%) used postoperative autologous blood salvage and reinfusion [26]. More widespread application of these techniques should be considered as part of optimal surgical blood conservation in CABG [18, 51, 52].

More recent evolving practices that may further minimize or eliminate homologous blood transfusion in CABG are preoperative autologous blood donation and acute normovolemic hemodilution, although guidelines and indications for the implementation of these interventions have not yet been adequately defined. The need for homologous blood in patients undergoing CABG who predeposit autologous blood has been shown to be reduced from 83% in patients who did not predonate autologous units to 27% in patients who predonated autologous blood units [17] and from 64% to 38%, respectively, in another series [16]. Idiopathic hypertrophic subaortic stenosis, aortic stenosis, congenital cyanotic heart disease, unstable angina or symptoms of disease on the day of donation, congestive heart failure, cardiac arrhythmias or atrioventricular block, and left main coronary artery disease should be considered high risk factors in autologous blood donors [53]. Although the overall safety of autologous blood donation using these guidelines has not been demonstrated by large-scale clinical studies, this intervention has been shown to be a highly effective method of reducing homologous blood transfusion needs in carefully selected patients [16, 17]. In one series, 97 (45%) of 217 first-time CABG procedures were accompanied by autologous blood predonation. This practice is currently underutilized in CABG, as demonstrated by a multicenter audit in which participation of eligible patients in autologous blood donation before primary, elective CABG occurred in only 18 (3.3%) of 540 patients [26].

Special blood requests such as designated blood (from donors known to the recipient) for CABG patients are also issues that need to be addressed. Designated blood transfusion is a controversial transfusion practice because of questions concerning the risk/benefit of designated versus homologous blood [54–58]. To date, the evidence indicates that designated blood is no safer, and may in fact be less safe, than homologous blood in terms of infectious risk [55, 56]. Recent reports [59, 60] indicate that a risk of graft versus host disease exists in patients undergoing CABG who are transfused with designated blood units of donors who are homozygous for HLA loci corresponding to recipients who are heterozygous for these HLA loci. The incidence of graft versus host disease has been estimated to be 1 in every 300 to 400 open heart operations in Japan [60]. Although the risk of this complication in the United States is unknown and appears to be low, the probability that blood homozygous for any extended haplotype will be given to recipients heterologous for the same extended haplotype has been estimated to be 1 in 500 for caucasians [61]. Routine irradiation of designated donor units from first-degree blood relatives has been recommended as a solution to this problem [62]. Patients undergoing CABG procedures should not be transfused with designated units from first-degree (parent-child or siblings) blood relatives; if this should become necessary, then these blood units should undergo gamma irradiation before transfusion.

Acute preoperative hemodilution has also been described as a safe and possibly effective technique to reduce homologous blood transfusion requirements in CABG [17, 18]. Acute hemodilution is the phlebotomy of whole (autologous) blood (2 to 5 U) in the operating suite just before operation, with normovolemic replacement with plasma volume expanders such as hydroxyethylstarch or balanced salt solutions. This technique results in less intraoperative RBC loss, as patients lose fewer RBCs for a given blood loss at lower hematocrits. The second advantage of this technique is that substantial amounts of autologous whole blood are available for transfusion needs. Possible limitations for use of this technique in patients undergoing CABG include preoperative anemia, unstable angina, impaired left ventricular function (ejection fraction < 0.5, cardiac index $< 2.5\ L \cdot m^{-2} \cdot min^{-1}$), left main coronary artery stenosis, ischemic electrocardiographic changes at rest, and impaired lung function [63]. This blood conservation intervention can also be considered to be underutilized; only 24 (4.4%) of 540 patients undergoing primary, elective CABG in a multiinstitution audit underwent acute preoperative hemodilution [26].

In addition to the technical interventions leading to the reduction of homologous blood transfusion, another important component to surgical blood conservation is acceptance of postoperative normovolemic anemia. The "transfusion-trigger" hematocrit has undergone reevaluation both in general surgery [64, 65] and in CABG [18, 38, 66], not only because of a general appreciation that oxygen transport is preserved at clinically significant levels of anemia, but also because of a renewed focus on issues of blood safety, such as risk of retrovirus disease transmission [4]. For example, one institution reported that 7% of hospitalized patients received one unit of blood during hospitalization, with 57% of these associated with cardiac operations, suggesting that single-unit transfusions may sometimes be appropriate [67]. Single-unit transfusion in patients undergoing CABG has also been described in a multiinstitutional survey [26]; of 498 patients without autologous blood available, 55 (11%) received one-unit transfusions and 75 (15%) received two-unit transfusions. Animal studies on the physiologic effects of acute anemia [68] indicate that more invasive monitoring techniques allowing calculation of oxygen extraction ratios and other physiologic values should help to define the transfusion trigger for individual patients [69]. These data and a recent report [70] indicate that in the setting of a conservative "transfusion-trigger," moderate application of additional blood conservation interventions, such as autologous blood predeposit, acute autologous hemodilution, and/or pharmacologic agents, will significantly reduce homologous blood transfusion in patients undergoing CABG. Interventions that could potentially reduce blood transfusion requirements in this setting include the use of deamino-D-arginine vasopressin for redo CABG [13] but not primary CABG [14], aprotinin [15], and fibrin glue [71] to reduce perisurgical blood loss. Another promising pharmacologic agent is recombinant human erythropoietin, shown to be effective in correcting the anemia of patients with renal failure [72] and the anemia associated with autologous blood donation before elective orthopedic operations [73]. The use of this agent in patients with poor tolerance for anemia, including patients undergoing CABG, could ameliorate the anemia associated with autologous blood donation as well as facilitate autologous RBC donation [74].

Guidelines for Reducing Homologous Red Blood Cell Transfusion

Based on the surgical, patient, and transfusion practice issues reviewed, guidelines for approaches to red blood cell transfusion in coronary artery bypass graft surgery are presented.

1. Institutions with CABG programs should establish protocols designed to reduce and minimize the need

for homologous RBC transfusion, especially for elective CABG procedures. These protocols should be developed through the collaboration of the hospital transfusion service (blood bank), the hospital transfusion committee, anesthesiology, the cardiovascular surgery program, and the regional blood center.

2. These protocols should incorporate a multifactorial approach to transfusion practice. The combination of a number of interventions (each of which possibly contributes a modest effect) can be anticipated to have a substantial impact on homologous blood requirements. Interventions proven to be safe and effective that should be used routinely include intraoperative and postoperative autologous blood salvage and reinfusion. An additional intervention that single institutions have shown to be safe and efficacious is preoperative autologous blood donation. Acute preoperative hemodilution and other promising pharmacologic interventions, such as recombinant human erythropoietin, aprotinin, deamino-D-arginine vasopressin, and fibrin glue, should be studied further. Fibrin glue used in this setting should preferably be autologous (patient's own) rather than derived from homologous blood because of the potential disease transmission risks of a homologous product.
3. Directed blood transfusion from first-degree blood relatives should be avoided. If this should become necessary, then these blood units should undergo gamma irradiation before transfusion.

Approach to the Coronary Artery Bypass Grafting Patient With Bleeding Complications

In the uncomplicated patient (usually elective patients undergoing primary procedures), blood components (plasma and platelets) need not be routinely given using the above guidelines. However, certain patients can be identified preoperatively who will likely need increased transfusion support [18, 19, 43].

Preoperative therapy should be directed toward specific bleeding disorders [75]. Evaluation of intraoperative bleeding can include clinical variables, such as estimates of blood loss, as well as intraoperative hematocrits for red cell transfusion needs [38]. The use of component therapy (platelets [9], fresh frozen plasma [10], and cryoprecipitate) is primarily based on these clinical variables, supplemented by laboratory measurements of platelet count, PT, PTT, bleeding time, and fibrinogen levels [31]. Previous studies have indicated that the prophylactic transfusion of platelets [76] or plasma [77] is not necessary and is therefore inappropriate [9, 10]. Excessive intraoperative bleeding or evidence of microvascular hemorrhage may identify patients who require intraoperative transfusion. Those requiring prolonged pump time or those receiving multiple units of intraoperative salvaged RBCs potentially require additional transfusion therapy [46, 78, 79]. Bleeding should also be closely followed in the postoperative period; excessive bleeding in the face of normal coagulation variables and platelet count suggests the need for surgical hemostasis. An approach to the management of diffuse (nonsurgical) bleeding in patients undergoing cardiac operations is presented in Figure 1.

Prompt laboratory evaluation in this setting can help to assess the need for the appropriate blood product support. These tests should include hematocrit, platelet count, PT, PTT, thrombin time, bleeding time, and fibrinogen levels. It should be emphasized that transfusions may have to be given based on clinical evaluation before laboratory data are available. However, laboratory assessment is essential for evaluating results and directing the therapy for persistent bleeding. Thrombocytopenia ($<100 \times 10^9$/L [100,000/μL]) in the setting of excessive or microvascular bleeding is an indication for platelet transfusion. Thrombocytopenia without evident hemorrhage is not by itself an indication for transfusion; conversely, a normal platelet count should not preclude platelet transfusions in the setting of microvascular bleeding if a qualitative platelet defect is demonstrated. The use of multiple red blood cell transfusions (3 to 4 units) does not necessitate routine (prophylactic) platelet transfusion [76].

Plasma transfusion therapy is indicated in the setting of suspected or documented multiple clotting factor deficiencies related to hemodilution or massive transfusion. One guideline for laboratory evidence for the transfusing of fresh frozen plasma is provided by PT and PTT greater than 1.5 times control [80]. In choosing a dose of fresh frozen plasma, it should be remembered that one unit in the average adult raises coagulation factors by only 3% [81]. In some patients, excessive bleeding may be caused by hypofibrinogenemia. This complication can occur in patients receiving large amounts of intraoperative or postoperative autologous blood salvage or massive transfusion [80]. Laboratory evidence of hypofibrinogenemia (<1.0 g/L [100 mg/dL]) indicates the need for cryoprecipitate therapy. One potential approach to patients with a high likelihood of requiring blood component therapy (eg, those undergoing reoperations [50] and the elderly [20]) is to harvest autologous platelet-rich plasma just before bypass, and then reinfuse after heparin neutralization [82]. However, indications for this procedure are yet to be established, because routine plasma or platelet administration is not necessary in patients undergoing elective primary CABG procedures [9, 10]. A benefit from transfusion of "fresh" (less than 12 hours old) blood is unproven [83, 84], and this practice is not justified based on a prospective randomized trial showing no benefit [84].

Guidelines for Blood Component Therapy in the Patient Undergoing Coronary Artery Bypass Grafting

1. No blood components should be given in the absence of clinically significant bleeding, irrespective of laboratory values. Plasma or platelet transfusions are not routinely needed as prophylaxis against bleeding in cardiac operations.
2. When excessive perioperative bleeding occurs, the decision to give platelets can be assisted by considering:

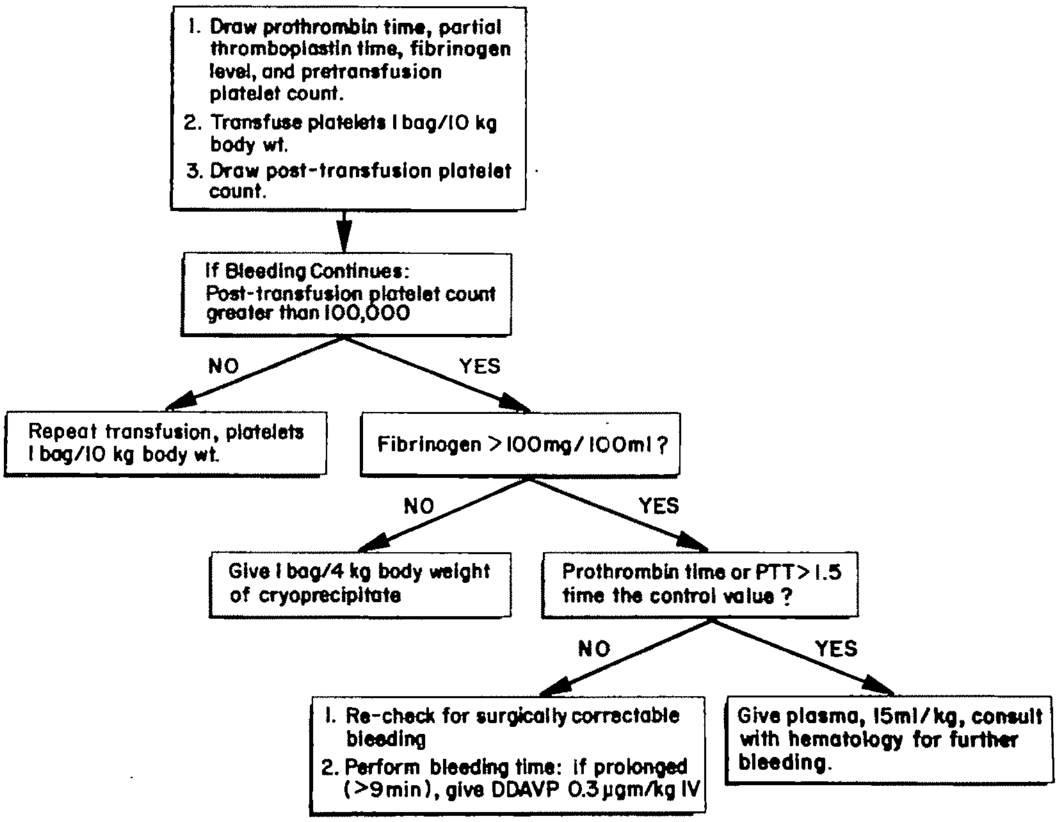
1. Draw prothrombin time, partial thromboplastin time, fibrinogen level, and pretransfusion platelet count.
2. Transfuse platelets 1 bag/10 kg body wt.
3. Draw post-transfusion platelet count.
If Bleeding Continues: Post-transfusion platelet count greater than 100,000
NO
YES
Repeat transfusion, platelets 1 bag/10 kg body wt.
Fibrinogen > 100mg/100ml ?
NO
YES
Give 1 bag/4 kg body weight of cryoprecipitate
Prothrombin time or PTT > 1.5 time the control value ?
NO
YES
1. Re-check for surgically correctable bleeding
2. Perform bleeding time: if prolonged (>9min), give DDAVP 0.3 μgm/kg IV
Give plasma, 15ml/kg, consult with hematology for further bleeding.

Fig 1. Management of postoperative bleeding in cardiac surgery patients: an algorithm approach. (DDAVP = *deamino-D-arginine vasopressin;* PTT = *partial thromboplastin time.)*

A. The clinical nature of the bleeding. Platelet abnormalities usually cause bleeding at other sites besides the operative field.
B. The platelet count. If the platelet count is unusually low (less than 50 × 10^9/L [50,000/μL]), transfusion is more likely to be helpful. Platelet counts should be used to guide platelet therapy in patients receiving large amounts of blood, when dilutional thrombocytopenia may accentuate the expected degree of platelet reduction.
C. The postoperative bleeding time. Prolongation to more than twice the normal range, when found more than 1 to 2 hours after operation, is good evidence for a clinically significant platelet abnormality if the platelet count is more than 100 × 10^9/L (100,000/μL).
D. Additional factors causing platelet dysfunction. Uremia or recent antiplatelet medications can contribute to the clinical picture of platelet-deficient hemorrhage.
E. Additional risks for bleeding. For example, patients with other coagulopathies, recent bleeding elsewhere, increased risk for hemorrhage at critical sites such as the central nervous system, or the need for reoperation may need more intensive blood component support than usual.

3. Investigation is being made of autologous platelet-rich plasma collected before and reinfused after cardiac bypass. Future studies may better define the benefits and appropriate patient candidates for this procedure. Because prophylactic plasma or platelet transfusion is unjustified in first-time elective CABG, this procedure cannot currently be recommended for routine CABG.
4. For support of intravascular volume, crystalloids or colloids should be used because they do not have the potential to transmit infection. Crystalloids are preferred because of their lower cost.
5. Plasma is indicated for demonstrated coagulation deficiencies, such as in massive transfusion (more than one patient blood volume) with documented coagulopathy, emergency reversal of warfarin when time does not permit correction with vitamin K (which can be accomplished in less than 8 to 10 hours), congenital clotting factor deficiencies, and multiple clotting factor deficiencies such as occur in liver disease and disseminated intravascular coagulation.

Conclusion

Most patients do well after a heart operation without any apparent evidence of immediate transfusion-related consequences. Follow-up studies of these patients have, however, contributed to our knowledge of the initial descriptions of transfusion-transmitted cytomegalovirus disease [85], the incidence of transfusion-associated non-A, non-B hepatitis [86], and the problem of leukoagglutination transfusion reactions as a cause of noncardiogenic pulmonary edema [87]. More recently, the probabilities of human immunodeficiency virus and type I human T-cell lymphotrophic virus transmission via blood components transfused in this patient population have been estimated [4], underscoring the importance of guidelines for transfusion practice in CABG.

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Notice From the American Board of Thoracic Surgery

The part 1 (written) examination will be held at the Hyatt-Regency, Dallas Fort Worth Airport, Dallas TX, on February 16, 1992. The closing date for registration is August 1, 1991.

To be admissible for the part II (oral) examination, a candidate must have successfully completed the part I (written) examination.

A candidate applying for admission to the certifying examination must fulfill all the requirements of the board in force at the time the application is received.

Please address all communications to the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Retrotracheal Mediastinal Goiter With Contralateral Extension

To the Editor:

I was interested in reading the case report written by Van Schil and associates [1]. No detail is given about the former cervical operation; it can be supposed it was performed on the left lobe. This occurrence would illustrate one of the traps of cervicothoracic surgery: the undiscovered plunge of a cervical goiter, when its "waistline" at the cervicothoracic opening is so thin it can be mistaken for a thick inferior thyroid pedicle and when its thoracic part is still small. As for the "crossing over" plunge, it happens more often from left to right because of anatomical vascular reasons and it may be anterior to the veins, in the thymic lodge, or posterior. This contralateral development can be suspected on a standard frontal chest roentgenogram: the tracheal clarity is lateral, but on the side where the cervical thyroid lobe is apparently enlarged (it would be on the right side in this case). Van Schil and associates do not mention this point and they do not give the time elapsed between the two operations.

It is remarkable that computed tomographic scans showed a substernal extension, as the thymic lodge looks quite empty on the lateral chest roentgenogram. The fibrous bands found at operation, secondary to the first operation, probably barred the goiter from the more natural and frequently used anterior way down the mediastinum. I personally think that two plain chest roentgenograms and an esophagram give sufficient information on a posterior mediastinal goiter. Frontal and lateral magnetic resonance imaging might be of interest. Thyroid scanning is negative in 45% of cases in my experience.

I agree with Van Schil and associates that sternotomy is better than blind operation in such a case. Posterolateral thoracotomy has also been used for a fixed, left behind, or undiagnosed thyroid mass. It can be misleading and it is generally very important to define the exact topography of any goiter before operation: it may happen that the larger cervical lobe does not plunge, while the contralateral, apparently normal lobe does, and that resection is therefore performed on the wrong lobe! Between 10% and 15% of patients with mediastinal goiters had a cervical thyroidectomy in the past and the mean delay between the two operations is more than 10 years. Usually symptoms of compression arise. Good chest roentgenograms and complete exploration of the two lobes are obligatory in cervical thyroid operations.

Posterior mediastinal goiters may have several extensions, dissociating trachea and esophagus. I never saw a posterior goiter crossing the mediastinum from the right; there is enough space on that side. But all forms can be observed (such as associated anterior and posterior extensions), and that is another argument against blind operation. Apart from plain frontal and lateral chest roentgenograms and esophagram, magnetic resonance imaging looks to me more promising than computed tomographic scans because of the possibility of frontal and lateral incidences; there are many traps in cervicomediastinal thyroid operations [2].

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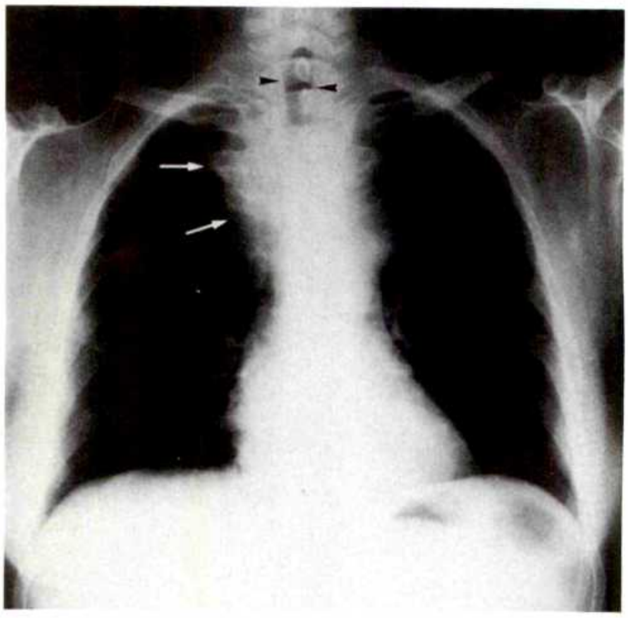

Fig 1. Anteroposterior chest roentgenogram showing right paratracheal mediastinal mass (arrows). *The tracheal clarity* (arrowheads) *is not displaced.*

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Reply

To the Editor:

I thank Dr Ribet for his very interesting remarks about mediastinal goiters. As the original report had to be shortened some details were omitted. The time interval between the two thyroid operations in our patient was 27 years. The first operation was performed at another hospital and no exact operative or pathological details were available. Presumably she underwent a bilateral subtotal thyroidectomy for a multinodular goiter.

On the standard frontal chest roentgenogram the tracheal clarity was not displaced laterally (Fig 1). On the lateral view the trachea was displaced ventrally by the posteriorally located mass but, of course, this did not give any information about contralateral extension. Computed tomographic and technetium thyroid scanning were not very helpful either in demonstrating the crossing over. Magnetic resonance imaging was not performed in our patient but looks very promising.

Considering the operative approach, a right lateral thoracotomy in this secondary goiter would not have offered any advantage as the right mediastinal thyroid came from the left side. A lateral thoracotomy may, however, be indicated in case of a primary intrathoracic goiter as blood supply entirely comes from local mediastinal vessels.

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Postirradiation Pericardiectomy

To the Editor:

I read the recent article by Ni and associates entitled "Futility of pericardiectomy for postirradiation constrictive pericarditis?" [1]. The problems experienced by them brought recall of a unique case seen by me several years ago. This patient unfortunately had the same poor outcome. The autopsy finding was also quite unexpected and of considerable importance.

The patient was a 38-year-old woman who had a diagnosis of Hodgkin's disease and was treated with mediastinal radiation therapy at the age of 23 years. She also had chemotherapy including Mustargen (mechlorethamine HCl), Oncovin (vincristine sulfate), and prednisone. She also underwent staging laparotomy. She had done well up until 5 months previously when progressive shortness of breath, precordial chest pressure, and ascites developed. She was diagnosed to have pericardial effusion, and failed to respond to intensive diuretic therapy. She subsequently underwent a pericardiocentesis, which was not productive. Through a left thoracotomy, a pericardial window was performed, and 500 mL of fluid drained. This was complicated by hypertension requiring ionotropic agents and fluid replacement. Microscopic description of the specimen was read as acute fibrinous and chronic pericarditis.

The patient remained quite symptomatic. She underwent cardiac catheterization, which demonstrated equalization of left and right atrial pressures, consistent with severe restriction.

A second procedure was initiated with a sternotomy incision. Pump oxygenator was available on standby. When the anterior mediastinum was entered, anatomical features were absent. Inspection of the anterior mediastinum demonstrated severe akinesis of the right ventricle. To allow further dissection and exposure of the pericardium, the patient was placed on femorofemoral bypass. A very extensive pericardiectomy was then performed, sharply removing a markedly thickened extremely fibrotic pericardium. An island was left to preserve the left phrenic nerve. Dissection was also performed to remove the thickened visceral pericardium wherever possible. The procedure was complicated by the previous pericardial window, which had produced considerable adhesions of the exposed lung to the heart. This resulted in local bleeding and air leak from the lung. The patient was weaned from cardiopulmonary bypass with ionotropic assistance.

The postoperative course was complicated by continual air leak from the mediastinum requiring persistent placement of a drainage tube. The patient was progressing in terms of stabilization of rhythm, cardiac function, and ambulation. On the ninth postoperative day she was found cyanotic and unresponsive and failed to respond to cardiac resuscitation.

Similar to the results of Ni and associates, this patient died. Autopsy examination revealed mesothelioma involving the pericardium, right pleura, and the posterior atrial septum. Although severe pericardial adhesions were expected, the findings at the surgical procedure were far more advanced than had been anticipated. The pericardium was markedly thickened and extremely tough and fibrotic. The difficulty in handling these adhesions is consistent with the fact that an unexpected malignancy was present.

Although the overall outcome may not have been improved, the option of cardiopulmonary bypass allowed a far more extensive removal of pareital pericardium [2]. Also, this allowed more extensive manipulation of the heart and removal of the visceral pericardium or epicardial layer. The advantage of use of cardiopulmonary bypass with pericardiectomy has been previously described, and bypass has been used by myself and my associates over the years [2].

The lessons learned by Ni and associates need to be taken into consideration. I also believe that in a setting of chemotherapy and radiation therapy the possible presence of malignancy should be considered in decisions regarding patient management.

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Efficacy of Pulmonary Artery Versus Left Ventricular Venting

To the Editor:

I have read with great interest the paper "Pulmonary artery versus left ventricular venting: a radioisotope study of left ventricular function" by Mitchell and associates [1]. The authors failed to show any differences in left ventricular function as measured by radionuclide techniques either immediately or late postoperatively. They concluded that pulmonary artery and left ventricular vents are equally satisfactory in terms of postoperative ventricular performance. I wonder whether the results of this study warrant those conclusions.

A detailed inspection of this paper reveals that every patient underwent aortic valve replacement for isolated aortic valve stenosis. All hearts were arrested by the infusion of cold St. Thomas Hospital cardioplegic solution through the aortic root.

In my experience, the reason why surgeons use a left ventricular vent in aortic valve replacement operations is to maintain a dry field to make the operation easier. It has already been demonstrated that flaccid hearts arrested by cardioplegic solutions do not benefit from a left ventricular vent as far as additional myocardial protection is concerned [2]. Also, the excision of the native aortic valve guarantees that intraventricular pressure could never exceed atmospheric pressure.

It is not surprising that no differences in postoperative left ventricular function were found between the two groups, as any kind of venting probably has no impact on myocardial protection in the described situation.

The mentioned mechanical damage secondary to left ventricular venting is possible but, in my experience, extremely rare. Regarding the incidence of air embolism, if pulmonary artery venting is as effective as left ventricular venting in emptying the ventricle, the presence of intracavitary air and potential source of embolization should be the same in both groups.

It would have been interesting to know if pulmonary artery venting was capable of producing as dry a field as left ventricular venting. Unfortunately, Mitchell and associates made no comment about this. This is important because that is the reason we all use a left ventricular vent in aortic valve replacements.

Anybody interested in determining the efficacy of pulmonary artery versus left ventricular venting should use a different clinical model. Surgeons routinely using fibrillatory arrest without aortic cross-clamping are in an enviable position to determine the efficacy of any kind of venting as, in these circumstances, left ventricular decompression is essential for myocardial protection [3]. Also, patients with severely calcified ascending aortas in whom surgeons are forced to use a "no-touch" technique, without aortic cross-clamping and forced fibrillatory arrest, provide a clinical opportunity to address this issue.

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Reply

To the Editor:

My colleagues and I thank Dr Garibaldi for his comments. We do not claim to have demonstrated any clear advantage to the use of pulmonary artery vents; rather, we suggest their use because of the apparent equality of function yet the absence of the disadvantages associated with left ventricular vents. These disadvantages include the risk of damage to the left ventricle and mitral valve. Although such injuries are uncommon, they have been well described and we see no reason to add to the risks of operation unnecessarily. Moreover, it has been suggested that in the presence of ischemic heart disease, focal dyskinesia may occur if the vent is inserted by direct puncture of the apex of the left ventricle [1], so giving rise to further reason for concern.

In our experience a pulmonary artery vent is equally as good as a left ventricular vent in maintaining a dry operative field. Although we agree that this is an important reason for venting during aortic valve replacement, we dispute the suggestion that it is the only reason. The contribution of the vent towards the prevention of rewarming, although small in this situation, must still be considered. More important, however, is the avoidance of distention during reperfusion, as this is well known to impair ventricular function [2]. Until the aortotomy is closed we accept that distention is unlikely to occur even in the absence of a vent. However, because of the systemic hypothermia used by most surgeons, full myocardial rewarming takes a considerable time and will not be complete before the aortotomy is closed. If ventricular function does not return immediately, then in the absence of a vent, distention can occur and will do so during the critical period of rewarming and reperfusion. We disagree with Dr Garibaldi and believe that venting in this situation does have an impact on myocardial protection and performance and therefore our study could indeed be expected to demonstrate a difference between different types of vents.

As regards air embolism, admittedly once in situ the two types of vent should remove intracavity air equally well. However, because the left ventricular vent is inserted into the systemic side of the circulation, there is a risk of air embolism associated with the actual placement and removal of the catheter. For obvious reasons this is not a problem with pulmonary artery venting.

Finally, although we would agree that the model of a patient with pure aortic stenosis is not ideal, we cannot conceive a better subject. We would dispute the model suggested by Dr Garibaldi, which is only applicable to closed procedures. Unfortunately, in practice, "closed procedures in adults" implies coronary artery operations. As it is impossible to accurately quantify the extent of the coronary artery disease and its effect on function, it would also be impossible to obtain two homogeneous and identical groups of patients to study; without doing so, however, the results would be hard to interpret. It is difficult to control all the possible variables, but ischemia is the one factor that will influence ejection fraction the most.

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Thymectomy for Myasthenia Gravis

To the Editor:

I am writing in reference to the article by Mulder and associates [1] describing their results of transsternal thymectomy in the treatment of myasthenia gravis and my subsequent letter to the Editor and Dr Mulder's reply [2, 3].

Doctor Mulder and I agree with the "logic of removing as much thymic tissue as possible," we both perform considerably more comprehensive dissections than many, and we agree that our results support the need for this type of extensive dissection. He believes, however, that the somewhat more extensive dissection I perform [4], especially in the neck, does not yield better results. He also believes the analysis of our data [5] is incorrect as we did not include 29 resections for myasthenia gravis without thymoma, 15 resections for myasthenia gravis with thymoma, and 8 reoperations following previous limited resections for recurrent or persistent myasthenia gravis.

My associates and I believe the analysis of our data was appropriate. We, as most, are trying to determine the results of thymectomy on patients with myasthenia gravis without the presence of thymoma or previous thymectomy. Although we did 124 consecutive "maximal" thymectomies between 1977 and 1985, the last 29 resections were not available for analysis at the time the study was undertaken and accordingly were not included in the analysis. Patients with thymoma in association with myasthenia gravis are known to have a significantly poorer prognosis. Accordingly, to include the 15 patients with thymoma in the analysis of patients undergoing thymectomy for myasthenia gravis without thymoma would be inappropriate. The 8 reoperations were not included in the analysis as they were a select group of patients who had failed previous thymectomy and represent an entirely different patient mix. In fact, some investigators believe they will not be helped by reoperation, although in our experience they are.

Accordingly, it was appropriate to exclude these three groups of patients, the comparison between our series and Dr Mulder's is valid, and the "maximal" procedure, which removes more thymus in many instances than Dr Mulder's operation, appears to give better results.

On the other hand, and most importantly, Dr Mulder and I are in complete agreement that whatever surgical technique is employed, extreme care must be taken to avoid injury (direct trauma or cautery) to both phrenic nerves, the left vagus nerve in the chest, and both recurrent laryngeal nerves in the neck even if in protecting these nerves some microscopic thymus is left behind.

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Reply

To the Editor:

Doctor Jaretzki and associates have offered their reasoning for excluding three groups of patients with myasthenia gravis rather than reporting their entire experience with "maximal" thymectomy. Our report contained *all* patients including those with thymomas, and some reoperations on patients who had no improvement after a cervical or possibly incomplete transsternal thymectomy, and even those operated on relatively recently who might be expected to improve with time—this latter group comprising almost one fourth of Jaretzki's entire series (29/124) being "not available for analysis" 2 to 3 years after operation.

In addition it would have seemed to us that a strong case could be made for "maximal" thymectomy if in the 8 patients undergoing reoperation for failed (incomplete?) thymectomy, residual thymic tissue had been found in aberrant locations (neck, aorticopulmonary window, etc) and these patients had shown improvement. To exclude them as a "select group of patients . . . [that] represent an entirely different patient mix" would seem inconsistent with his subsequent sentence stating that "some investigators believe they will not be helped by reoperation, although in our experience they are."

Doctor Jaretzki quite correctly reminds us that great care must be taken to not injure the phrenic, vagus, and recurrent laryngeal nerves as these injuries are especially devastating in the patient with myasthenia gravis. Such injuries are most likely to occur while dissecting in the neck.

As mentioned in our previous response, Dr Jaretzki and associates have made a substantial contribution with their cadaver studies showing the aberrant locations in which thymic tissue has been found. It may be that for those occasional patients with severe residual or recurrent myasthenia in whom one has done the transsternal, "complete" thymectomy that we have described, a transsternal reexploration with a concomitant cervical incision and exploration could be considered while recognizing the increased morbidity associated with this. We remain unconvinced that such a "maximal" thymectomy should be considered the procedure of choice for all patients with myasthenia gravis.

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Intraoperative Photodynamic Therapy for Malignant Mesothelioma

To the Editor:

We read with interest the article by Watts and colleagues entitled "Giant benign mesothelioma" [1], and we would like to bring to your attention a new approach that is being investigated at our institution for the possible management of diffuse pleural malignant mesothelioma. This approach involves the preoperative delivery of a photosensitizer (dihematoporphyrin ether) and debulking of disease to 5 mm thickness, either by pleurectomy or extrapleural pneumonectomy, followed by intraoperative delivery of 630-nm light using an argon pump-dye laser. This therapy, known as photodynamic therapy, is based on the in vivo selective retention of sensitizer by malignant tissue and relatively superficial depth penetration of light wavelengths (5 to 7 mm), which may be useful in the treatment of surface malignancies (neoplasms of skin, bladder, bronchus, and esophagus) [2].

We recently treated a 58-year-old man with right-sided diffuse pleural mesothelioma by performing an extrapleural pneumonectomy 2 days after he received 2.0 mg/kg of dihematoporphyrin ether. After the diaphragm was replaced with dura mater, the only areas of gross residual disease included the lateral chest wall at the diaphragmatic sulcus, and these areas were debulked to 5 mm thickness or less. After hemostasis was obtained, seven sterile photodiodes were sewn to the chest cavity superiorly (1), inferiorly (2), medial, lateral, and posteriorly (1 each), and one photodiode was secured to the right ventricular epicardium. These photodiodes were then attached to a continuously integrating computerized photometer for instantaneous and cumulative recording of light power density and integrated energy density from the areas monitored by the photodiodes. The chest cavity was then filled with 0.02% intralipid for light scattering, and pleural illumination was accomplished with two specially instrumented laser fibers, one each from two lasers. Seven watts of continuous 630-nM light was used to deliver a total dose of 15 J/cm^2 to the chest cavity. The heart was treated to a total dose of 11 J/cm^2. After light delivery the photodiodes were removed, and the chest was closed without drainage. Total operative time was 8 hours, with 37 minutes of laser time.

The patient was extubated the following morning. The only evidence of substantial chemical abnormality included a rise in the serum creatinine level from 90 to 140 μmol/L (1.0 to 1.6 mg/dL) on the night of operation, which normalized with hydration over the next 48 hours, and a rise in the creatine kinase level from a baseline of 44 to 11,030 U/L 72 hours after photodynamic therapy. Myoglobin was absent from the urine (<15 ng/L). Fractionation of the creatine kinase revealed 100% muscle band, and by the time of discharge on the ninth postoperative day, the total creatine kinase level was 369 U/L. Examination of the specimen revealed areas of intense pink fluorescence when illuminated with 514-nM light, and the tumor was a mixed epithelial-fibrous mesothelioma. The patient was instructed to avoid direct sunlight for 5 weeks, and he was seen in follow-up

2 and 4 months after the procedure. There have been no oxygen requirements, all wounds have healed per primum, and a baseline chest computed tomogram reveals only the right hydrothorax. Serum creatine kinase level has normalized and the patient has returned to work.

It is probably true that with advanced malignant mesothelioma, the majority of cases explored and resected will have residual disease. The use of photodynamic therapy in this context assumes that the bulk of the intrapleural disease can be resected, leaving discontinuous patches of macroscopic or microscopic tumor that have previously accumulated the sensitizer and that can be ablated by 630-nM light. Theoretically, this approach is feasible because thoracic malignancies, specifically mesothelioma [3], will respond to photodynamic therapy in vitro and in vivo (Feins RH, Hilf R, Ross H, Gibson S, unpublished data). For use of intrapleural photodynamic therapy in humans, of course, the maximum tolerated dose of the treatment must be determined, and this is underway in a phase I trial at our institution. Naturally, it is too early to comment on efficacy; however, we thought that thoracic and medical oncologists would be interested in this technique, which offers an innovative approach in these unfortunate patients.

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Respiratory Function After Thoracic Operations

To the Editor:

Three hundred one patients underwent thoracotomy within a period of 1 year in our department of general thoracic surgery (ZKH Gauting). The average age of our patients was 51 years, with a range from 12 to 81 years. Twenty-two patients were older than 70 years. Bronchial carcinoma was the most common diagnosis (37%). Other diagnoses were: bronchial carcinoma, 111 (37%); spontaneous pneumothorax, 45 (15%); chondroma/tuberculoma, 39 (13%); empyema, 35 (12%); mycetoma, 11 (4%); pleuromesothelioma, 8 (3%); carcinoid, 6 (2%); and other, 46 (15%). The most common operation was lobectomy. Other operations were: lobectomy, 86 (29%); pleurodesis, 50 (17%); enucleation, 45 (15%); wedge resection, 38 (13%); decortication, 35 (12%); pneumonectomy, 15 (5%); sleeve resection, 7 (2%); other, 25 (8%). All our patients were instructed before operation in respiratory physical therapy. This included instruction in coughing and deep breathing as well as in the use of an incentive spirometer. Those patients suffering from obstructive disease were treated by bronchial dilators that were continued postoperatively. Values of forced expiratory volume in 1 second were assessed before and 14 days after operation in all patients.

Comparison of preoperative and 14 days postoperative values of forced expiratory volume in 1 second revealed a decrease of 0.5 to 0.6 L in all patients. No significant differences were found for the decrease of forced expiratory volume in 1 second between patients who underwent pneumonectomy, lobectomy, or enucleation (Table 1). This suggests that the operative trauma is more important in decreasing respiratory function after thoracic operation than the type of resection performed. It confirms the need for extensive respiratory therapy under adequate analgesic management.

Table 1. Preoperative and Postoperative Values of Forced Expiratory Volume in 1 Second[a]

Operation	Preoperative (L)	Postoperative (L)
Pleurodesis/enucleation	2.7 ± 0.76	2.2 ± 0.58
Lobectomy	2.6 ± 0.66	2.0 ± 0.52
Pneumonectomy	2.4 ± 0.62	1.8 ± 0.43

[a] Values are shown as mean ± standard deviation.

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Brief notices of meetings, courses, and symposia relevant to cardiothoracic surgery are published as a service. They should be formatted as follows: meeting title, date, place, sponsor, accreditation, director, fee, minimum/maximum attendance, and address/phone for further information.

Announcements should be sent at least 4 months in advance of publication to *The Annals of Thoracic Surgery*, 3108 Queeny Tower, Barnes Hospital Plaza, St. Louis, MO 63110-1041; telephone, (314) 361-6084.

American College of Surgeons, San Francisco, California—October 7–12, 1990

For information on this meeting contact American College of Surgeons, 55 E Erie St, Chicago, IL 60611; or telephone (312) 664-4050.

Fifty-sixth Annual Scientific Assembly, American College of Chest Physicians, Toronto, Ontario, Canada—October 22–26, 1990

This meeting is sponsored by the American College of Chest Physicians and the International Academy of Chest Physicians and Surgeons. For information, contact the American College of Chest Physicians, 911 Busse Highway, Park Ridge, IL 60068; or telephone (708) 698-2200.

Thirty-seventh Annual Meeting of the Southern Thoracic Surgical Association, Cerromar Beach, Puerto Rico—November 8–10, 1990

For information on this meeting, contact Gordon F. Murray, MD, Southern Thoracic Surgical Association, 111 East Wacker Dr, Chicago, IL 60601-4301; or telephone (312) 644-6610 (facsimile: (312) 938-1214).

Sixty-third Scientific Session of the American Heart Association, Dallas, Texas—November 12–15, 1990

For information on this meeting, contact American Heart Association, 7320 Greenville Ave, Dallas, TX 75231; or telephone (214) 750-5300.

Global Plenum on Organ Transplantation, Lucknow, India—December 14–16, 1990

For information on this meeting, contact Prof Probal Ghosh, Plenum Secretariat, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Raebareli Rd, Post Box 375, Lucknow 226001, India.

Cardiopulmonary Rehabilitation: Status '91, Gainesville, Florida—January 28–30, 1991

For information on this meeting, telephone Michael L. Pollock, PhD, at (904) 392-9575.

Twenty-seventh Annual Meeting of The Society of Thoracic Surgeons, San Francisco, California—February 17–20, 1991

For information on this meeting, contact Richard P. Anderson, MD, The Society of Thoracic Surgeons, 111 East Wacker Dr, Chicago, IL 60601-4301; or telephone (312) 644-6610.

Fortieth Annual Meeting of the American College of Cardiology, Atlanta, Georgia—March 3–7, 1991

For information on this meeting, contact American College of Cardiology, 9111 Old Georgetown Rd, Bethesda, MD 20814; or telephone (800) 253-4636.

Seventy-first Annual Meeting of The American Association for Thoracic Surgery, Washington, DC—May 6–8, 1991

For information on this meeting, contact American Association for Thoracic Surgery, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Quality of Life After Open Heart Surgery, Antwerp, Belgium—May 16–18, 1991

This meeting is sponsored by The World Health Organization, the European Society of Cardiology, and the International Society and Federation of Cardiology. For information on this meeting, contact P. J. Walter, MD, FACS, Professor and Chairman of the Department of Cardiovascular Surgery, University Clinic of Antwerp, B 2520 Antwerp, Wilrijkstraat 10, Belgium; or telephone 00323.829.11.11 (facsimile: 00323.830.20.99).

Coronary Surgery: Clinical and Basic Perspectives, Sheffield, England—May 17, 1991

For information on this meeting, contact G. D. Angelini, Clinical Sciences Centre, Northern General Hospital, Herries Rd, Sheffield S5 7AU, England.

Fifth International Symposium on Cardiac Bioprostheses, Avignon, France—May 24–27, 1991

This meeting is sponsored by the Scientific Committee for the Advancement of Cardiac Bioprostheses. For information on this meeting, contact Beverly Farrier, ProMedica International, 620 Newport Center Dr, Suite 575, Newport Beach, CA 92660.

Ninth World Symposium on Cardiac Pacing and Electrophysiology, Washington, DC—May 28–31, 1991

This meeting will incorporate the Twelfth Annual Scientific Session of the North American Society of Pacing and Electrophysiology. For information on this meeting, contact IXth World Symposium Secretariat, c/o NASPE, 377J Elliot St, Newton Upper Falls, MA 02164; or telephone (617) 237-1866 (facsimile: (617) 431-1991).

The International Society for Cardiovascular Surgery/The Society for Vascular Surgery, Boston, Massachusetts—June 3–5, 1991

For information on this meeting, contact the Association offices, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Seventeenth Annual Meeting of The Western Thoracic Surgical Association, Seattle, Washington—June 26–29, 1991

For information on this meeting, contact The Western Thoracic Surgical Association, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

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Please reply to Box 350. 350F/K

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Please respond to Box 354. 354G/L

Thoracic surgeon wanted: A busy thoracic practice 20 miles south of Boston seeks to add a third surgeon by July 1991. Candidate must be board certified or eligible. This active practice operates from a multihospital referral base. The community is an ideal area for family living, with excellent schools and nearby Boston and Cape Cod cultural and recreational facilities. Competitive salary, malpractice insurance, and excellent benefit package. Please respond with CV.

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Cardiovascular/thoracic surgeon, BC/BE, to join well-established private practice on Florida west coast. Expertise in adult cardiac, vascular, and thoracic surgery required. Recent graduate with transplant experience preferred. Excellent compensation and benefits leading to full partnership. Please include curriculum vitae and references with response.

Please respond to Box 363. 363G/J

Cardiothoracic surgeon, BE/BC, to join established 1-man private practice involved with adult cardiac, thoracic, and vascular surgery in major Midwest community. New or recent graduate preferred. Salary leading to partnership. Please send CV, clinical experience, and references.

Please respond to Box 366. 366H/K

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Please respond to Box 368. 368H/J

Cardiac surgeon, board certified or board eligible, for very busy private practice group doing primarily adult cardiac and vascular surgery. Seeking an Associate to join well-established practice in northeast Ohio metropolitan area. Please respond with curriculum vitae and references.

Please respond to Box 370. 370H/A

Cardiovascular surgeon: We are looking for a recently trained B/E or B/C CV surgeon to join our very busy and enjoyable 2-man cardiovascular department at The Billings Clinic, a progressive major regional multispecialty clinic in the northern Rocky Mountain region. This is an outstanding practice opportunity for the right individual seeking a challenging and rewarding surgical practice in surroundings conducive to the highest quality of living for the surgeon and his or her family.

For further information, please call or send CV to Hewes D. Agnew, MD, or J. Scott Millikan, MD, The Billings Clinic, PO Box 35100, Billings, MT 59107-5100; (406) 256-2500. 375H/J

Thoracic surgeon, BC/BE, needed to join two others in a busy thoracic surgical practice without open heart surgery. Located in a prime suburb of NYC with a good quality of life. Rapid partnership advancement. Recent graduate preferred. Please reply with a curriculum vitae.

Please respond to Box 376. 376H/J

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Please respond to Box 377. 377H/J

Cardiovascular/thoracic surgeon to complete 3-man group in adult cardiothoracic private practice in Michigan. Must be board eligible or certified. References required.

Please respond to Box 382. 382H/J

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Please respond to Box 384. 384H/K

Cardiothoracic surgeon (BE/BC) to join newly established practice of primarily adult cardiac surgery. Expanding practice is now ready for second surgeon. Excellent opportunity for a recent graduate in the beautiful area of Virginia.

Please respond to Box 390. 390I/J

Cardiothoracic surgeon, ABTS certified/eligible, with primary interest in cardiac surgery, wanted immediately to replace member of established surgical group practicing in Allentown, PA. Competitive salary/fringe benefits. Position leads to partnership for successful candidate.

Please send curriculum vitae, clinical experience, and references to Joan M. Persa, Administrator, PO Box 8575, Allentown, PA 18105-8575; (215) 821-8790. 393I/B

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Thoracic, vascular, and general surgeon to join a busy, all boarded, private practice on eastern Long Island. Should be board certified or eligible. Excellent salary and benefits, leading to a full partnership. Send curriculum vitae.

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Cardiovascular surgeon to become second surgeon in new (two year old) adult cardiac program on west coast of Florida.

Submit CV to Manatee Cardiac Surgeons, PA, 201 4th Ave East #2, Bradenton, FL 34208. 396I/K

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For full details contact Wendy Perlman (800) 327-1585. 397I/J

Cardiovascular surgeon, board certified/eligible, wanted to join two surgeons in an established cardiovascular program in the southeastern USA. Must have experience in adult cardiac, thoracic, and vascular surgical procedures. Salary/full partnership negotiable. Please send resume.

Please respond to Box 398. 398I/B

Cardiovascular and thoracic surgeon: Excellent opportunity for a board certified/eligible cardiovascular and thoracic surgeon to join in a new, growing department in a metropolitan New York community on Long Island. Academic/private practice setting with excellent compensation. 533-bed community teaching hospital. Experience in electrophysiologic surgery, artificial devices, and/or transplantation helpful. Please respond with curriculum vitae.

Please respond to Box 403. 403I/L

Thoracic/vascular surgeon needed to join prominent, active 3-man group in desirable suburban community in greater NY metropolitan area. Excellent opportunity for well-trained, highly motivated individual. Recent graduate preferred. Please respond with CV.

Please respond to Box 404. 404J/C

Congenital cardiothoracic surgery: The Children's Hospital of New Jersey is seeking a pediatric cardiothoracic surgeon to join its well-established program. Candidates should be board certified by the American Board of Thoracic Surgery and have 2 to 3 years experience in congenital cardiac surgery at fellowship and/or practice level. We are looking for a candidate with excellent surgical skills and a strong interest in research and teaching. The position carries an academic appointment at The University of Medicine and Dentistry of New Jersey–The New Jersey Medical School, which has an accredited program in Thoracic Surgery.

Send CV to Joseph V. Cotroneo, MD, The Children's Hospital of New Jersey/United Hospitals Medical Center, 15 South Ninth St, Newark, NJ 07107; (201)268-8671 (8606). 405J/K

Chief, Pediatric Cardiovascular Surgery: The Ohio State University Department of Surgery, Division of Thoracic and Cardiovascular Surgery, and the Columbus Children's Hospital are seeking an experienced pediatric cardiovascular surgeon to serve as Chief of Pediatric Cardiovascular Surgery at Columbus Children's Hospital in Columbus, Ohio. Preference will be given to persons with extensive experience in all aspects of pediatric cardiovascular surgery, especially neonatal corrections. Applicants should be board certified in thoracic surgery with specialized training and experience in pediatric cardiovascular surgery. The successful applicant will be Chief of the Department of Pediatric Cardiovascular Surgery at Columbus Children's Hospital, a 315-bed dedicated children's facility in Columbus, Ohio, and a full-time faculty member within the Division of Thoracic and Cardiovascular Surgery at The Ohio State University. In addition to clinical expertise, a strong interest in teaching is mandatory. The development of an active research program is desirable and a new modern research facility, the Wexner Institute for Pediatric Research, is available for that purpose.

Please send CV with names and addresses of three references to P. David Myerowitz, MD, Karl P. Klassen Professor and Chief, Division of Thoracic and Cardiovascular Surgery, The Ohio State University Hospitals, N-825 Doan Hall, 410 West 10th Ave, Columbus, OH 43210. The Ohio State University is an affirmative action/equal opportunity employer. 406J

Adult cardiac, peripheral vascular, and thoracic surgery practice in Midwest with rapidly growing referral base desires well-qualified surgeon for association leading to partnership. Practice includes busy transplant program. Academic affiliation available. Send CV and three references.

Please respond to Box 407. 407J/L

Thoracic and cardiovascular surgeon, board certified or eligible, wanted for an established practice in the Midwest with experience in adult cardiac, thoracic, and peripheral vascular surgery. Experience or training in heart/lung transplantation preferred. Initial contact should include curriculum vitae, clinical experience, and names of references.

Please respond to Box 408. 408J/B

Cardiovascular and thoracic surgeon—board eligible or qualified—wanted to join 4 physicians in very active private practice in greater Chicago area. Respond with curriculum vitae.

Please respond to Box 409. 409J/K

Vacancy exists for a BE/BC thoracic and cardiovascular surgeon. Candidate must have proven clinical and laboratory abilities and vascular surgery fellowship. Training required in major complex vascular procedures.

Please forward curriculum vitae to Carolyn G. Gray, 2002 Holcombe Blvd, Houston, TX 77030; or telephone (713) 795-7463. EOE. 410J/K

Retired or retiring cardiovascular surgeons willing to assist only in surgery. No calls.

Please respond to Box 411. 411J

Cardiovascular and thoracic surgeons wanted for growing practice in the Southwest. Applicant must be ABTS eligible or certified. Send curriculum vitae.

Please respond to Box 412. 412J

Assistant surgeon—Busy adult and pediatric cardiac and vascular surgery group in suburban Washington, DC, is seeking a non-board certified surgeon, preferably with cardiovascular experience, to assist in the operating room. Excellent salary and fringe benefits.

Please respond with CV to Virginia Heart Surgery Associates, 3301 Woodburn Rd, Suite 301, Annandale, VA 22003. 413J/L

An academic position in thoracic and cardiovascular surgery is available at SUNY–Health Science Center at Syracuse. The position primarily involves adult and pediatric cardiac surgery; however, experience in all other aspects of thoracic surgery is required. A commitment to teaching and research is necessary. This is an excellent opportunity for an individual interested in academic pursuits as well as an excellent clinical experience. The academic appointment will be based on the credentials of the applicant. SUNY–Health Science Center at Syracuse is an EEO/AA employer.

Please send curriculum vitae to Frederick B. Parker, Jr, MD, Professor & Chairman, Department of Surgery, and Chief, Cardiopulmonary Division, SUNY–Health Science Center, 750 E Adams St, Syracuse, NY 13210. 414J

Cardiac surgeon—board certified/eligible to be third member of established practice in suburb of Washington, DC, expanding to second hospital. Excellent compensation and benefits. Great opportunity for recent graduate wishing to locate in area of nation's capital. Practice focus is primarily in adult cardiac surgery with additional work in thoracic and peripheral vascular surgery available.

Please send curriculum vitae to PO Box 34945, Bethesda, MD 20817. 415J/C

Cardiovascular surgeon (BC/BE) to join 2 recently trained, board certified associates in a well-established adult cardiac surgery program in a multispecialty clinic affiliated with a stable referral system of 24 satellite clinics. Active program includes arrhythmia surgery. Midwest location is 30 minutes from the Minnesota lakes district.

Contact Dakota Medical Center, 1702 South University Dr, Fargo, ND 58103; Dr John Hicks, Director, Recruiting Division, (701) 280-3338. 416J/C

Cardiovascular research scientist: The Division of Cardiothoracic Surgery, University of South Florida, seeks a cardiovascular scientist with a PhD in Physics/Bioengineering and expertise in myocardial mechanics and function. Proven research productivity and ability to obtain grant support is required. The successful applicant will be an integral part of an expanding cardiovascular research program.

Respond to Dr P. McKeown, #730, 4 Columbia Dr, Tampa, FL 33606. 417J

Cardiovascular and thoracic surgeon wanted to join a busy, expanding practice in the western suburbs of Chicago. Immediate position available for BC/BE individual. Please forward CV with letter of introduction.

Please respond to Box 418. 418J

Cardiothoracic/vascular surgeon: board certified in general and thoracic surgery. Minimum of 2 years of practice experience. Very active private 5-surgeon multispecialty practice. Located in western Pennsylvania. Salary leading to full partnership. Excellent benefits. Detailed CV must accompany response. All replies treated confidentially.

Please respond to Box 419. 419J/C

BC/BE general/thoracic surgeon—California: We need a general surgeon who is also trained in thoracic surgery and interested in an excellent practice doing general and thoracic surgery (no open heart). We offer the opportunity to establish your own private practice with no investment, guaranteed income. Well-equipped 112-bed full-service hospital. A growing area with new businesses and stable economy. Our service area population is now 90,000. Located in central California near Sequoia National Park. Tulare offers an excellent family-oriented lifestyle with all expected amenities. Beautiful homes, located close to hospital and office, are affordably priced. Good schools, many community activities, and abundant recreation including golf, tennis, skiing, mountain and equestrian activities. Easy access to all California's major metropolitan and resort areas.

Please contact Tulare District Hospital Physician Recruiting Office, PO Box 90112, Los Angeles, CA 90009; (800) 468-2687. 420J

Cardiothoracic surgeon—to join a 2-man private practice in cardiac, thoracic, and vascular surgery in the Great Lakes region. Must be board certified or eligible. Recent graduates considered. Send curriculum vitae.

Please respond to Box 421. 421J/C

Cardiothoracic surgeon needed to join established 2-man group in practice of adult cardiac, pulmonary, and vascular surgery. Must be board certified or eligible and graduate from a US medical school.

Please respond to R. W. Ruess, MD, 1834 Broadway, Cape Girardeau, MO 63701. 422J/C

Cardiovascular-thoracic surgeon—BE/BC to join established and growing 2-surgeon group in northeastern US. Practice includes high volume cardiac and general thoracic surgery. Recent trainee preferred. Submit CV.

Please reply to Box 423. 423J/C

SITUATIONS WANTED

Cardiothoracic surgeon, 36, 2 years' experience in adult cardiac surgery. Excellent results. Wishes to relocate to West Coast or the Northwest.

Please respond to Box 369. 369H/J

Cardiovascular and thoracic surgeon, 45, ABS, ABTS, NE university trained, seeks relocation. Experienced, publications, academic appointment.

Please respond to Box 386. 386I/K

Cardiovascular and thoracic surgeon, 52, looking for a salaried position in cardiac surgery or serving as assistant surgeon. ABS, ABTS certified. Experienced in every facet of the field. May start immediately.

Please respond to Box 391. 391I/J

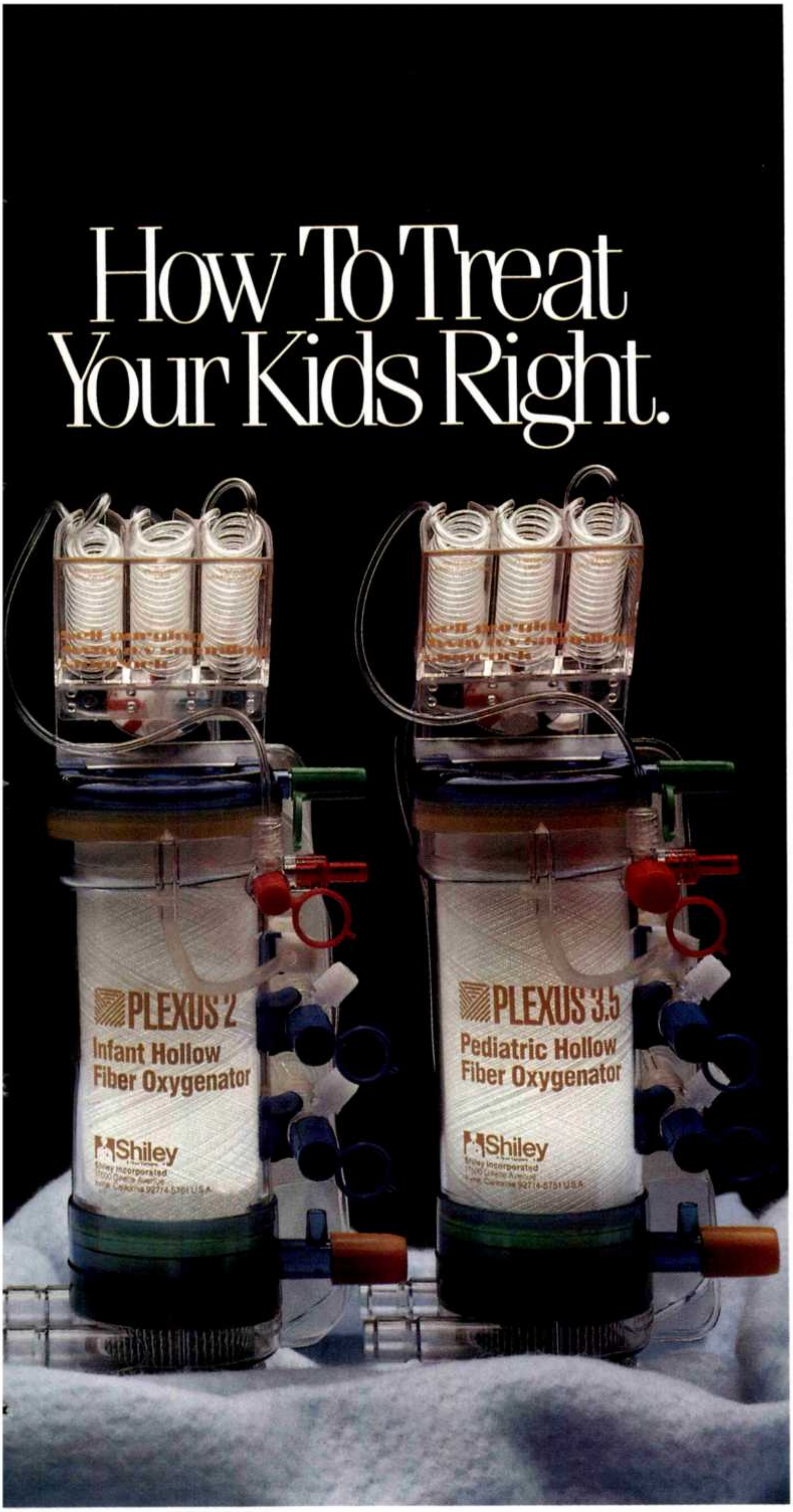
FELLOWSHIPS

Fellowship in cardiovascular surgery for 1 year available January 1 or July 1 with group doing approximately 1,000 cardiac procedures per year and large volume of vascular procedures. ABS eligible or certified, California medical license required or readily obtainable. Ideal for a person between general surgery residency and thoracic surgery residency.

Please respond with curriculum vitae to Bradley J. Harlan, MD, 5301 F Street, Suite 312, Sacramento, CA 95819. 364G/L

Clinical cardiac surgical fellowship at Massachusetts General Hospital, Boston, Massachusetts: One-year appointment beginning January 1, 1991, or July 1, 1991. Salary commensurate with training and consistent with hospital scale. Intensive exposure and experience opportunity in cardiac surgery. PG-5 or beyond. Interviews and references required.

For additional information, please write Mortimer J. Buckley, MD, Chief, Cardiac Surgery, Massachusetts General Hospital, Boston, MA 02114. 385I/J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Interested parties send CV to:

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Phone: 502-825-7470

The Veterans Affairs Medical Center, Jackson, Mississippi is recruiting a Board Certified General or Thoracic surgeon to serve as Chief, Surgical Service. The incumbent will be responsible for coordinating the activities of 26 surgical residents and 17 full/part time staff physicians. The Jackson VA Medical Center is a 455-bed acute care medical center with a 120-bed nursing home care unit. There are 126 surgical beds, including a 12-bed Surgical Intensive Care Unit. Activation of a new seven-room operating suite, including ambulatory surgery, is scheduled for April 1991. The Jackson VA Medical Center is directly adjacent to the University of Mississippi Medical Center and is an integral part of its teaching programs. Applicants are expected to play a major role in these programs and must be eligible for faculty appointment. Interested applicants should send curriculum vitae to:

Roland B. Robertson, M.D.
Chief of Staff
VA Medical Center
1500 E. Woodrow Wilson Drive
Jackson, MS 39216-5199

The deadline for application is February 1, 1991

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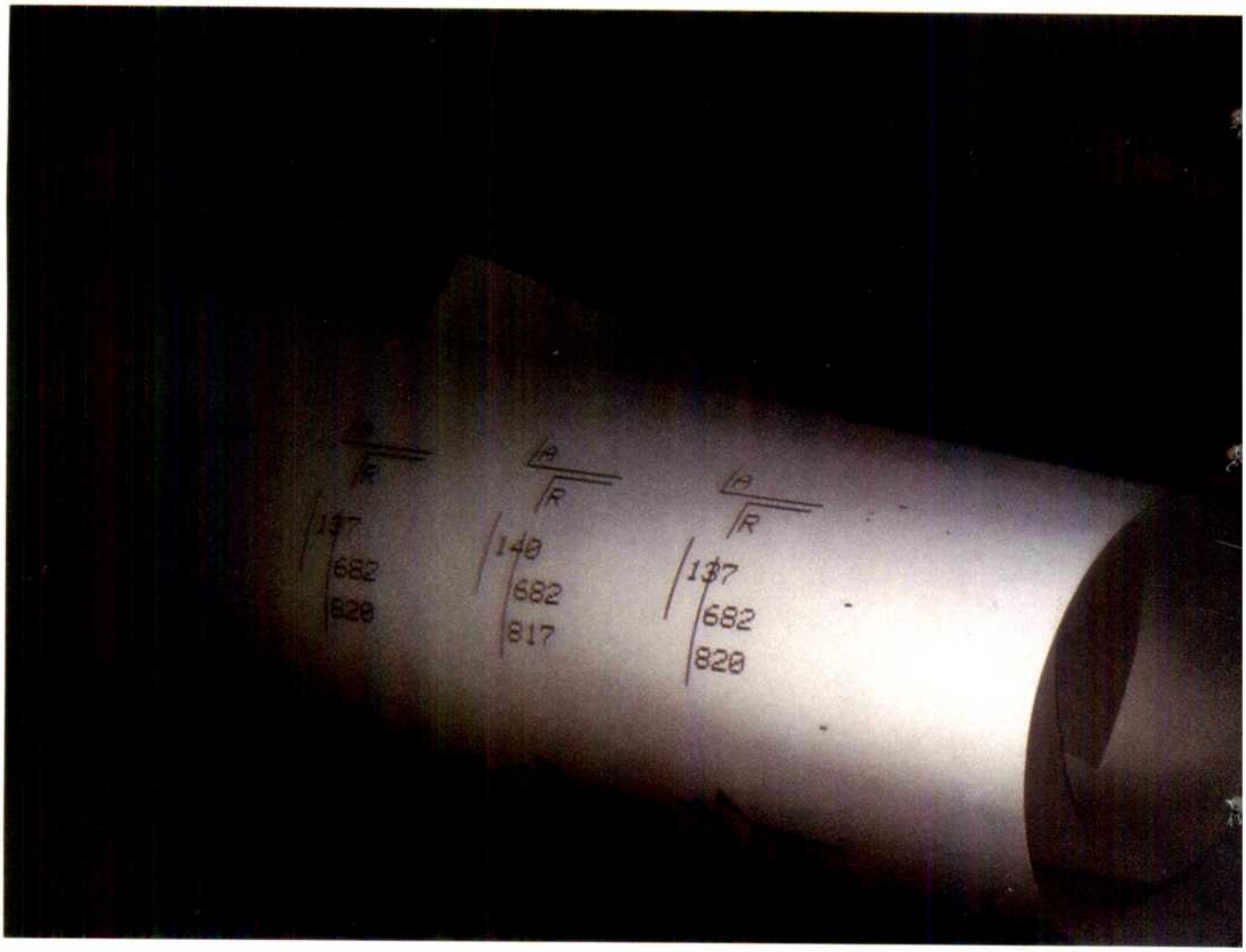

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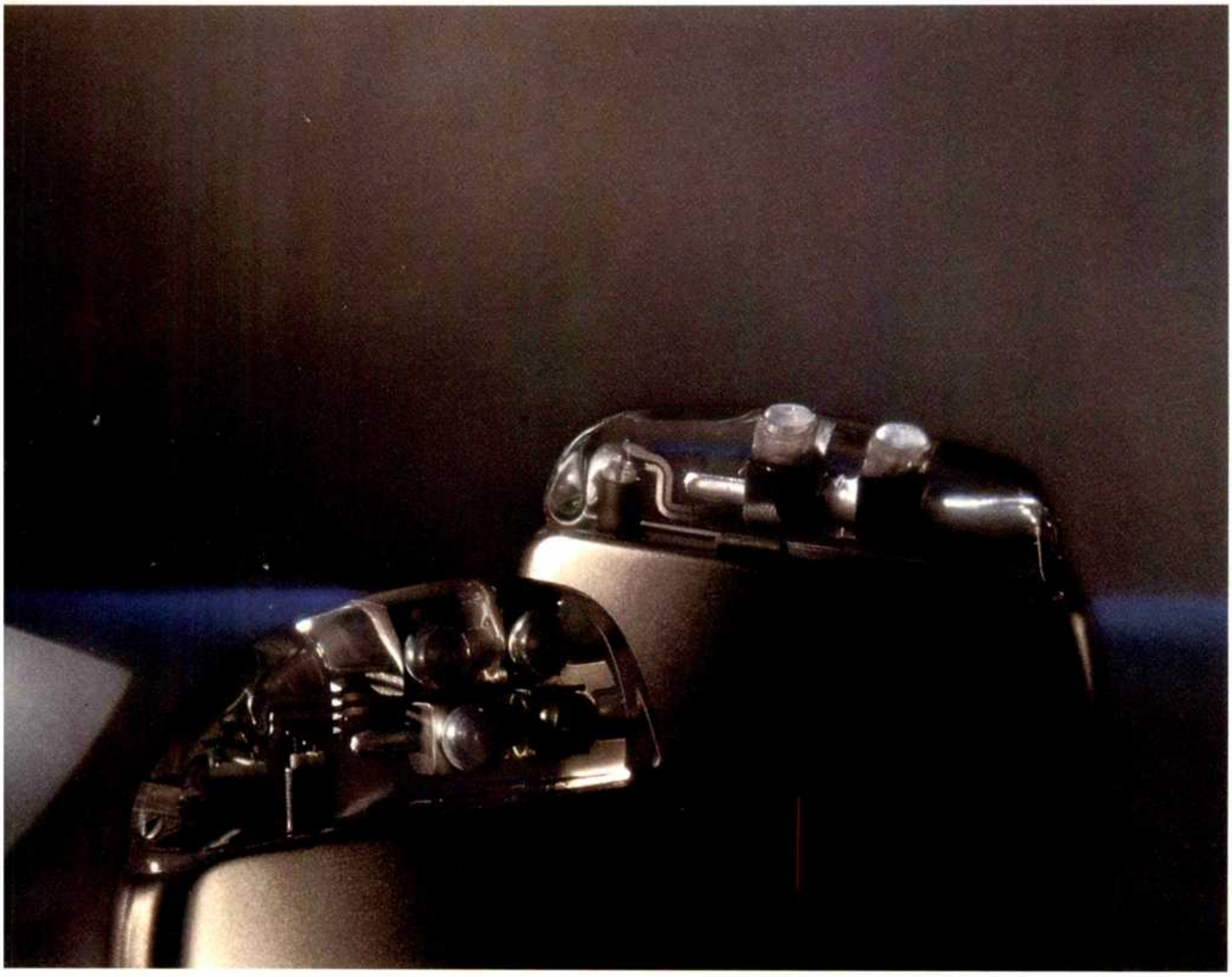

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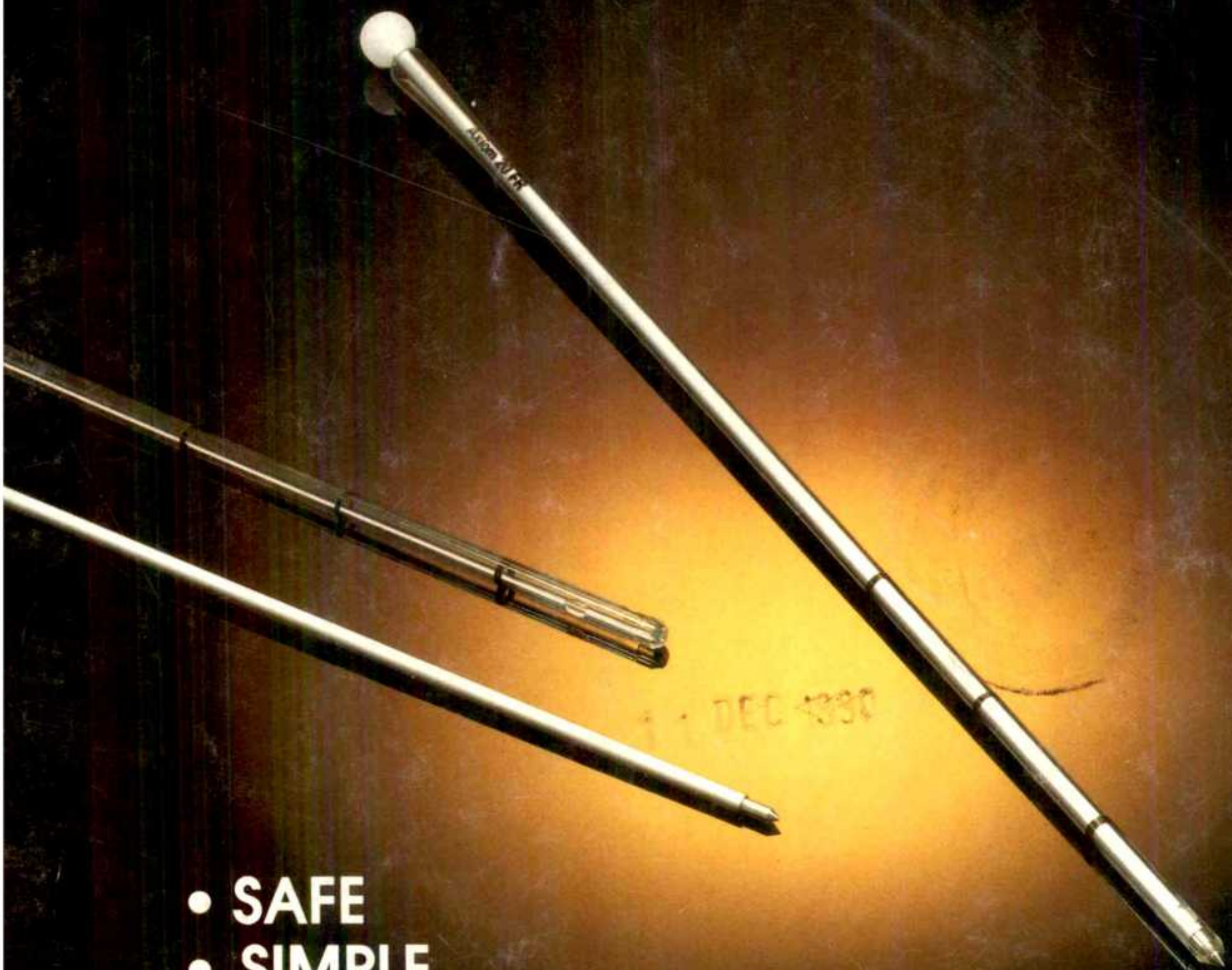

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Zinacef* **(sterile cefuroxime sodium, Glaxo)** **Zinacef*** **(cefuroxime sodium injection)** **BRIEF SUMMARY**

The following is a brief summary only. Before prescribing, see complete prescribing information in Zinacef* product labeling.

INDICATIONS AND USAGE: Zinacef* is indicated for the treatment of patients with infections caused by susceptible strains of the designated organisms in the following diseases:

1. **Lower Respiratory Tract Infections,** including pneumonia, caused by *Streptococcus pneumoniae* (formerly *Diplococcus pneumoniae*), *Haemophilus influenzae* (including ampicillin-resistant strains), *Klebsiella* spp, *Staphylococcus aureus* (penicillinase- and non-penicillinase-producing), *Streptococcus pyogenes*, and *Escherichia coli*.
2. **Urinary Tract Infections** caused by *E coli* and *Klebsiella* spp.
3. **Skin and Skin Structure Infections** caused by *S aureus* (penicillinase- and non-penicillinase-producing), *S pyogenes*, *E coli*, *Klebsiella* spp, and *Enterobacter* spp.
4. **Septicemia** caused by *S aureus* (penicillinase- and non-penicillinase-producing), *S pneumoniae*, *E coli*, *H influenzae* (including ampicillin-resistant strains), and *Klebsiella* spp.
5. **Meningitis** caused by *S pneumoniae*, *H influenzae* (including ampicillin-resistant strains), *Neisseria meningitidis*, and *S aureus* (penicillinase- and non-penicillinase-producing).
6. **Gonorrhea:** Uncomplicated and disseminated gonococcal infections due to *Neisseria gonorrhoeae* (penicillinase- and non-penicillinase-producing strains) in both males and females.
7. **Bone and Joint infections** caused by *S aureus* (including penicillinase- and non-penicillinase-producing strains).

Clinical microbiological studies in skin and skin structure infections frequently reveal the growth of susceptible strains of both aerobic and anaerobic organisms. Zinacef has been used successfully in these mixed infections in which several organisms have been isolated. Appropriate cultures and susceptibility studies should be performed to determine the susceptibility of the causative organisms to Zinacef.

Therapy may be started while awaiting the results of these studies; however, once these results become available, the antibiotic treatment should be adjusted accordingly. In certain cases of confirmed or suspected gram-positive or gram-negative sepsis or in patients with other serious infections in which the causative organism has not been identified, Zinacef may be used concomitantly with an aminoglycoside (see PRECAUTIONS). The recommended doses of both antibiotics may be given depending on the severity of the infection and the patient's condition.

Prevention: The preoperative prophylactic administration of Zinacef may prevent the growth of susceptible disease-causing bacteria and thereby may reduce the incidence of certain postoperative infections in patients undergoing surgical procedures (eg, vaginal hysterectomy) that are classified as clean-contaminated or potentially contaminated procedures. Effective prophylactic use of antibiotics in surgery depends on the time of administration. Zinacef should usually be given one-half to one hour before the operation to allow sufficient time to achieve effective antibiotic concentrations in the wound tissues during the procedure. The dose should be repeated intraoperatively if the surgical procedure is lengthy.

Prophylactic administration is usually not required after the surgical procedure ends and should be stopped within 24 hours. In the majority of surgical procedures, continuing prophylactic administration of any antibiotic does not reduce the incidence of subsequent infections but will increase the possibility of adverse reactions and the development of bacterial resistance.

The perioperative use of Zinacef has also been effective during open heart surgery for surgical patients in whom infections at the operative site would present a serious risk. For these patients it is recommended that Zinacef therapy be continued for at least 48 hours after the surgical procedure ends. If an infection is present, specimens for culture should be obtained for the identification of the causative organism and appropriate antimicrobial therapy should be instituted.

CONTRAINDICATIONS: Zinacef* is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

WARNINGS: BEFORE THERAPY WITH ZINACEF* IS INSTITUTED, CAREFUL INQUIRY SHOULD BE MADE TO DETERMINE WHETHER THE PATIENT HAS HAD PREVIOUS HYPERSENSITIVITY REACTIONS TO CEPHALOSPORINS, PENICILLINS, OR OTHER DRUGS. THIS PRODUCT SHOULD BE GIVEN CAUTIOUSLY TO PENICILLIN-SENSITIVE PATIENTS. ANTIBIOTICS SHOULD BE ADMINISTERED WITH CAUTION TO ANY PATIENT WHO HAS DEMONSTRATED SOME FORM OF ALLERGY, PARTICULARLY TO DRUGS. IF AN ALLERGIC REACTION TO ZINACEF OCCURS, DISCONTINUE THE DRUG. SERIOUS ACUTE HYPERSENSITIVITY REACTIONS MAY REQUIRE EPINEPHRINE AND OTHER EMERGENCY MEASURES.

Pseudomembranous colitis has been reported with the use of cephalosporins (and other broad-spectrum antibiotics); therefore, it is important to consider its diagnosis in patients who develop diarrhea in association with antibiotic use.

Treatment with broad-spectrum antibiotics alters normal flora of the colon and may permit overgrowth of clostridia. Studies indicate a toxin produced by *Clostridium difficile* is one primary cause of antibiotic-associated colitis. Cholestyramine and colestipol resins have been shown to bind the toxin *in vitro*.

Mild cases of colitis may respond to drug discontinuation alone. Moderate to severe cases should be managed with fluid, electrolyte, and protein supplementation as indicated.

When the colitis is not relieved by drug discontinuation or when it is severe, oral vancomycin is the treatment of choice for antibiotic-associated pseudomembranous colitis produced by *C difficile*. Other causes of colitis should also be considered.

PRECAUTIONS: Although Zinacef* rarely produces alterations in kidney function, evaluation of renal status during therapy is recommended, especially in seriously ill patients receiving the maximum doses. Cephalosporins should be given with caution to patients receiving concurrent treatment with potent diuretics as these regimens are suspected of adversely affecting renal function.

The total daily dose of Zinacef should be reduced in patients with transient or persistent renal insufficiency (see DOSAGE AND ADMINISTRATION), because high and prolonged serum antibiotic concentrations can occur in such individuals from usual doses.

As with other antibiotics, prolonged use of Zinacef may result in overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Broad-spectrum antibiotics should be prescribed with caution in individuals with a history of gastrointestinal disease, particularly colitis.

Nephrotoxicity has been reported following concomitant administration of aminoglycoside antibiotics and cephalosporins.

Drug/Laboratory Test Interactions: A false-positive reaction for glucose in the urine may occur with copper reduction tests (Benedict's or Fehling's solution or with Clinitest* tablets), but not with enzyme-based tests for glycosuria (eg, Tes-Tape*). As a false-negative result may occur in the ferricyanide test, it is recommended that either the glucose oxidase or hexokinase method be used to determine blood plasma glucose levels in patients receiving Zinacef.

Cefuroxime does not interfere with the assay of serum and urine creatinine by the alkaline picrate method.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Although no long-term studies in animals have been performed to evaluate carcinogenic potential, no mutagenic potential of cefuroxime was found in standard laboratory tests.

Reproductive studies revealed no impairment of fertility in animals.

Pregnancy: ***Teratogenic Effects: Pregnancy Category B:*** Reproduction studies have been performed in mice and rabbits at doses up to 60 times the human dose and have revealed no evidence of impaired fertility or harm to the fetus due to cefuroxime. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed.

Nursing Mothers: Since Zinacef is excreted in human milk, caution should be exercised when Zinacef is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in children below 3 months of age have not been established. Accumulation of other members of the cephalosporin class in newborn infants (with resulting prolongation of drug half-life) has been reported.

ADVERSE REACTIONS: Zinacef* is generally well tolerated. The most common adverse effects have been local reactions following intravenous administration. Other adverse reactions have been encountered only rarely.

Local Reactions: Thrombophlebitis has occurred with intravenous administration in 1 in 60 patients.

Gastrointestinal: Gastrointestinal symptoms occurred in 1 in 150 patients and included diarrhea (1 in 220 patients) and nausea (1 in 440 patients). Onset of pseudomembranous colitis symptoms may occur during or after antibiotic treatment (see WARNINGS).

Hypersensitivity Reactions: Hypersensitivity reactions have been reported in less than 1% of the patients treated with Zinacef and include rash (1 in 125). Pruritus, urticaria, and positive Coombs' test each occurred in less than 1 in 250 patients, and, as with other cephalosporins, rare cases of anaphylaxis, erythema multiforme, and Stevens-Johnson syndrome have occurred.

Blood: A decrease in hemoglobin and hematocrit has been observed in 1 in 10 patients and transient eosinophilia in 1 in 14 patients. Less common reactions seen were transient neutropenia (less than 1 in 100 patients) and leukopenia (1 in 750 patients). A similar pattern and incidence were seen with other cephalosporins used in controlled studies.

Hepatic: Transient rise in SGOT and SGPT (1 in 25 patients), alkaline phosphatase (1 in 50 patients), LDH (1 in 75 patients), and bilirubin (1 in 500 patients) levels has been noted.

Kidney: Elevations in serum creatinine and/or blood urea nitrogen and a decreased creatinine clearance have been observed, but their relationship to cefuroxime is unknown.

In addition to the adverse reactions listed above that have been observed in patients treated with cefuroxime, the following adverse reactions and altered laboratory tests have been reported for cephalosporin-class antibiotics:

Adverse Reactions: Toxic epidermal necrolysis, fever, vomiting, abdominal pain, colitis, vaginitis including vaginal candidiasis, toxic nephropathy, hepatic dysfunction including cholestasis, aplastic anemia, hemolytic anemia, hemorrhage.

Several cephalosporins have been implicated in triggering seizures, particularly in patients with renal impairment when the dosage was not reduced (see DOSAGE AND ADMINISTRATION). If seizures associated with drug therapy should occur, the drug should be discontinued. Anticonvulsant therapy can be given if clinically indicated.

Altered Laboratory Tests: Prolonged prothrombin time, pancytopenia, agranulocytosis, thrombocytopenia.

DOSAGE AND ADMINISTRATION: (See complete prescribing information in Zinacef* product labeling.)

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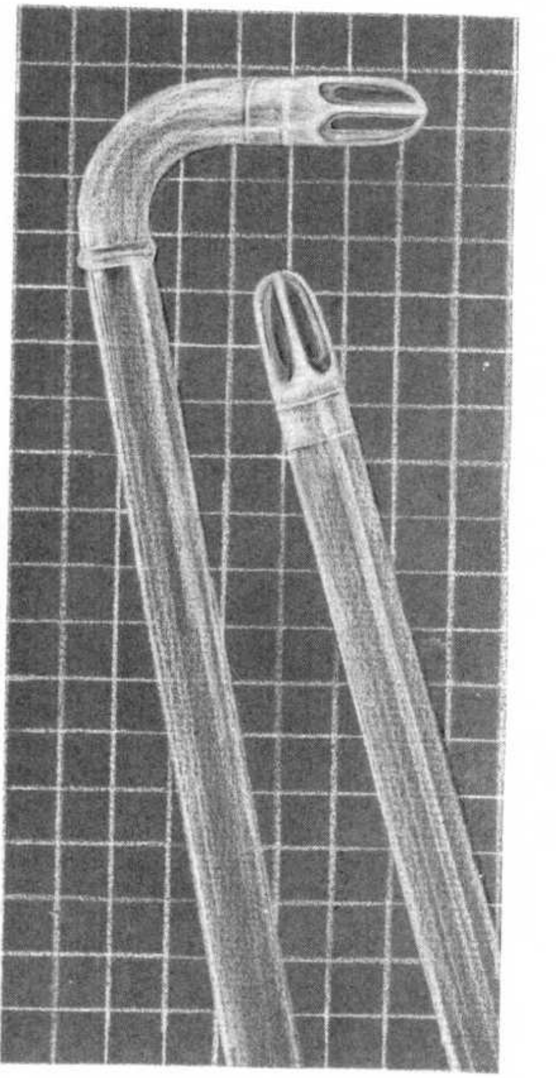

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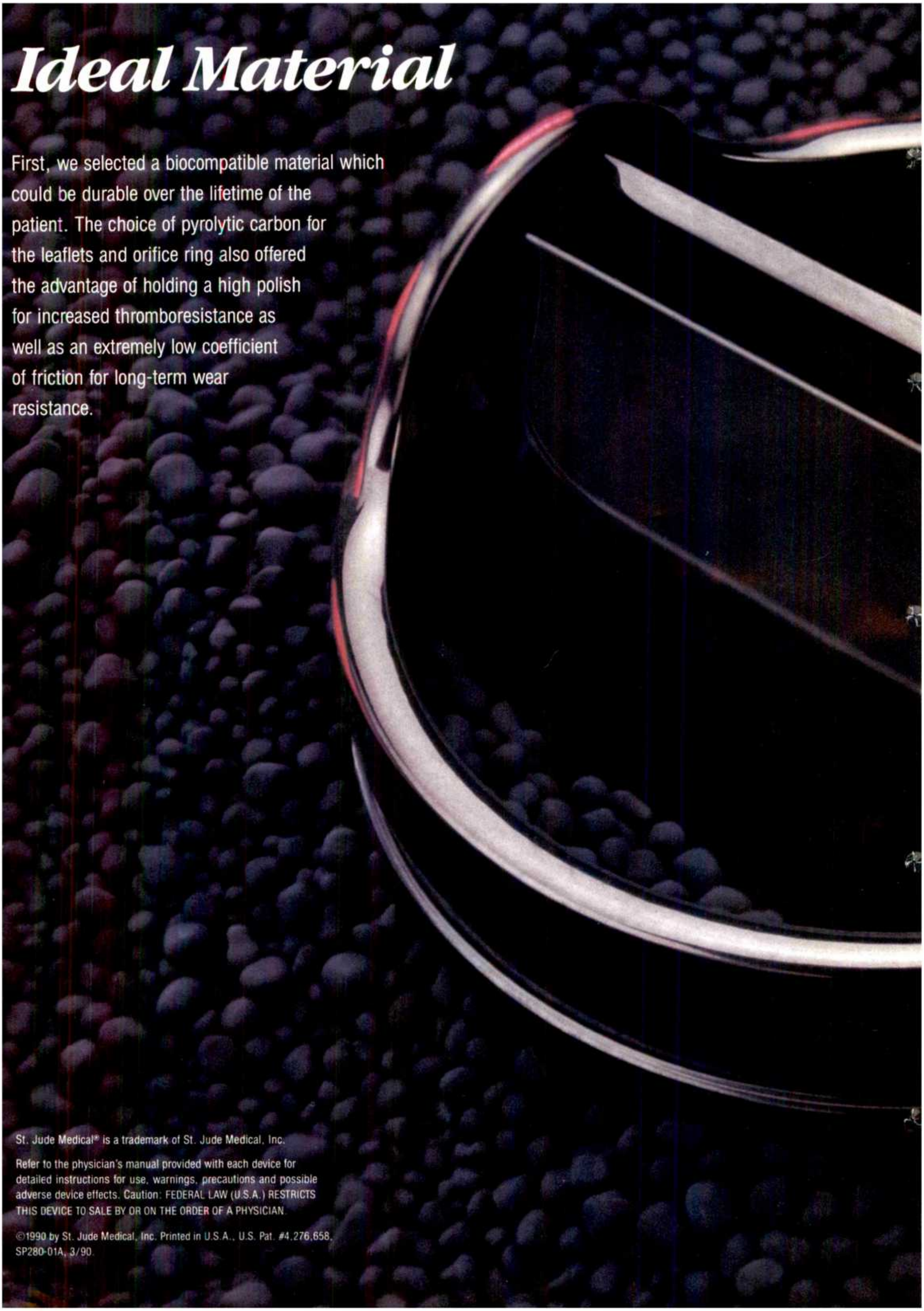

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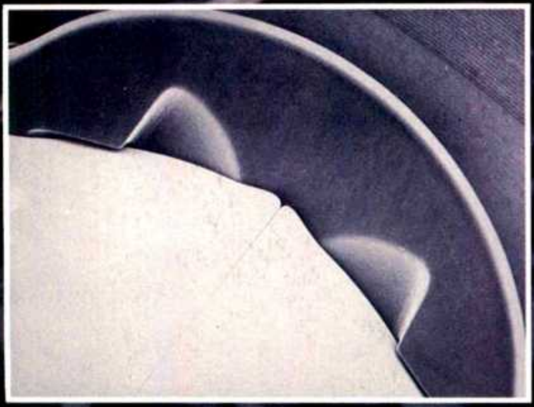

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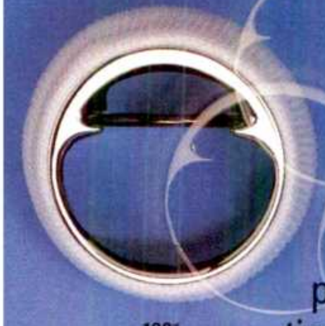

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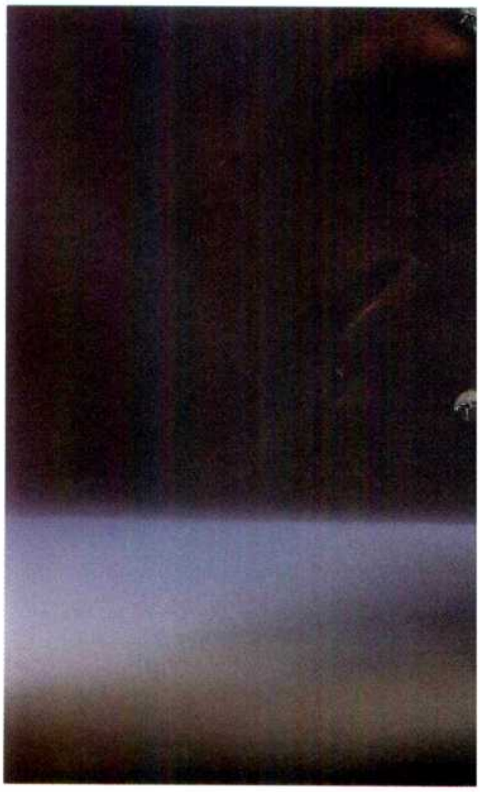

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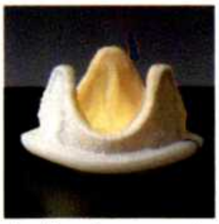

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Indications: Bioprostheses are indicated for use in patients suffering from valvular heart disease. Aortic and mitral valvular heart disease are conditions involving any of the following: obstructions of the heart valves, or stenosis; leakage of the valves, known as regurgitation or insufficiency; and combinations of the two, sometimes referred to as mixed disease or combined lesions. Valvular heart disease may be caused by any number of factors, including congenital abnormalities, infection by various microorganisms, degenerative calcification, rheumatic heart disease and rupture or dysfunction of the mitral subvalvular mechanism secondary to myocardial ischemia. Bioprostheses are used particularly in those patients for whom long-term anticoagulation is contraindicated or who may be difficult to maintain on anticoagulation therapy. The Carpentier-Edwards bioprosthesis is intended for use in patients whose aortic or mitral valvular disease is sufficiently advanced to warrant replacement of their natural valve with a prosthetic one. It is also intended for use in patients with a previously implanted aortic or mitral valve prosthesis which is no longer functioning adequately and requires replacement. In the latter case, the previously implanted prosthesis is surgically excised and replaced by the replacement prosthesis. **Warnings:** For Single Patient Use Only In the presence of conditions affecting calcium metabolism or when calcium-containing chronic drug therapies are used, the use of a mechanical prosthesis as an alternative should be considered. This is also true in patients under 20 years of age, in patients on a high calcium diet, in the presence of severe systemic hypertension, and in patients who are on maintenance hemodialysis. The decision to use a tissue valve must ultimately be made by the physician on an individual basis after a careful evaluation of the short- and long-term risks and benefits to the patient are made, and alternative methods of treatment are considered. Overall durability, especially long-term, has not been established for bioprostheses. Serious complications, sometimes leading to replacement of the valve and/or death, may be associated with the use of prosthetic valves (see Complications). A full explanation of the benefits and risks should be given to each prospective patient before surgery. **Note:** Bioprostheses should be used with caution in the presence of severe systemic hypertension or when the anticipated patient longevity is longer than the known longevity of the prosthesis. Careful and continuous medical follow-up (at least by an annual visit to the physician) is advised so that valve-related complications, particularly those related to material failure, can be diagnosed and properly managed. Recipients of prosthetic heart valves who are undergoing dental procedures should receive prophylactic antibiotic therapy to minimize the possibility of prosthetic infection. Bioprosthetic heart valve recipients should be maintained on anticoagulant therapy, except where contraindicated, during the initial healing stages after implantation, approximately 2 to 3 months. Anticoagulants should then be discontinued over a period of 10 days, except in those patients for whom indefinite anticoagulant protection is indicated, i.e., in the absence of sinus rhythm and in patients with a dilated left atrium, calcification of the atrial wall or history of previous atrial thrombosis. The appropriate anticoagulation therapy must be determined by the physician on an individual basis. **Adequate rinsing with physiological saline, as described in the Technique section, is mandatory before implantation to reduce the glutaraldehyde concentration. No other solutions, drugs, chemicals, antibiotics, etc. should ever be added to the glutaraldehyde or rinse solutions, as irreparable damage to the leaflet tissue, which may not be apparent under visual inspection, may result. The Carpentier-Edwards bioprostheses must be kept moist at all times. Drying out will cause irreversible damage to the leaflet tissue. To prevent drying out during implantation, the valves should be irrigated periodically on both sides with sterile physiological saline.** Passing diagnostic catheters and transvenous pacing leads across a Carpentier-Edwards bioprosthesis could damage the valve. Consequently, it is recommended that these procedures be avoided. Both the valve and glutaraldehyde storage solution are **sterile.** The outside of the jar is **not sterile** and must **not** be placed in the sterile field. Furthermore, the valve and container should **never** be subjected to sterilization procedures involving ethylene oxide, propylene oxide, steam, or irradiation. Storage between 10°C and 25°C is recommended; however, care should be exercised to avoid freezing or extreme heat, which may damage the valvular tissue. **Any valve that has been frozen or exposed to temperatures greater than 60°C should not be used for human implantation.** Glutaraldehyde may cause irritation of the skin, eyes, nose, and throat, and may also cause skin sensitization. Avoid prolonged or repeated contact or prolonged breathing of the vapor. Use only with adequate ventilation. In the event of contact, immediately flush the affected area with water. In the event of contact with eyes, seek medical attention. **Surgical Precautions:** **1.** When choosing a valve for a given patient, the size, age, and physical condition of the patient in relation to the size of the prosthesis must be taken into consideration to minimize the possibility of obtaining a suboptimal hemodynamic result. The selection of a valve, however, must ultimately be made by the physician on an individual basis after carefully weighing all of the risks and benefits to the patient. **2.** Due to the relative flexibility of the frame, care must be exercised to avoid the use of too large a prosthesis to prevent folding or extreme deformation of the stent that may render the valve incompetent. Oversizing may create highly localized mechanical stresses resulting in tissue failure in the form of detachment of the leaflets or porcine aortic wall from the valve stent posts. **3.** Left ventricular perforation or ulceration has been reported. Both open and closed chest cardiac massage in patients with an open strut mitral prosthesis increases the risk of ventricular perforation. Additionally, lifting, flexing, and manual compression of the distended heart after discontinuation of bypass may cause a commissure strut to be pushed through the endocardium, producing a dissecting hematoma with subsequent uncontrolled hemorrhage. **4.** The spacing of the sutures in the remnant of the valvular orifice and the prosthesis suture ring must be carefully matched to avoid folding of the leaflets or extreme distortion of the orifice. Edwards CVS Division has received reports in which individual mattress sutures, spanning a distance of 10 to 15mm, produced a purse-string effect causing compression of the valve orifice. **5.** When using interrupted sutures, it is important to cut the sutures close to the knots and to ensure that exposed suture tails will not come into contact with the leaflet tissue. Cases have been reported in which bioprostheses developed severe regurgitation and had to be replaced as a result of wear due to contact with sutures. **6.** Unlike rigid mechanical valves, the stent wall is soft and will not resist needle penetration. Accordingly, extreme care must be exercised when placing sutures through the sewing margin to avoid penetration of the side wall of the stent and possible laceration of the leaflet tissue. **7.** As with all prostheses that have open cages, free struts, or commissure supports, care must be exercised, particularly in mitral replacement, to avoid looping or catching a suture around the free legs which would interfere with proper valvular function. **8.** The stent of the bioprosthesis is asymmetrical to conform to the natural shape of the porcine aortic valve. Consequently, the commissure supports are not equidistantly spaced. The contrasting suture markers in the sewing ring of the mitral valve denote the greatest intercommissural distance. These two commissure supports should be oriented so as to straddle the ventricular outflow tract to avoid murmurs due to blood flow turbulence. **Note:** The contrasting suture material should not be removed from the prosthesis. **9.** In the aortic position, the struts should correspond to the remnants of the natural commissures so as not to obstruct the coronary ostia. **10.** A serial number tag is attached to the sewing ring of each valve by a suture. This serial number should be checked against the number on the jar; if any difference is noted, the valve should be returned unused. This tag should not be detached from the valve until implant is imminent. Care should be exercised to avoid cutting or tearing the suture ring cloth during removal. **11.** Gentle handling is required for all implantable devices. If the valve is dropped, damaged, or mishandled in any way, it must not be used for human implantation. **Complications:** As with all prosthetic heart valves, serious complications, sometimes leading to death, may be associated with the use of tissue valves. In addition, complications due to individual patient reaction to an implanted device, or to physical or chemical changes in the components, particularly those of biological origin, may occur at varying intervals (hours or days) necessitating reoperation and replacement of the prosthetic device. Complications associated with the use of Carpentier-Edwards bioprostheses include stenosis, regurgitation through an incompetent valve, perivalvular leak, endocarditis, hemolysis, thromboembolism, thrombotic obstruction, bleeding diatheses related to the use of anticoagulant therapy, ventricular perforation by stent posts, looped sutures, and malfunctions of the valve due to distortion at implant or physical or chemical deterioration of valve components. Types of tissue deterioration include infection, calcification, thickening, perforation, suture abrasion, instrument trauma, and leaflet or aortic wall detachment from the valve stent posts. These complications may present clinically as abnormal heart murmur, shortness of breath, exercise intolerance, dyspnea, orthopnea, anemia, fever, arrythmia, hemorrhage, transient ischemic attack, stroke, paralysis, low cardiac output, pulmonary edema, congestive heart failure, cardiac failure, and myocardial infarct. **Note:** Based on reports in the literature on tissue valves, there appears to be an increased incidence of leaflet calcification in patients under the age of 20. In this regard, animal research studies show that a high systemic calcium level can lead to early calcification. Furthermore, at least one published report describes an apparent causal relationship between the consumption of daily calcium supplements and early leaflet calcification in an adult. When feasible, repeated intravenous injections containing calcium should be avoided during the postoperative period; and excessive milk or dairy product consumption should be avoided in children. There are no clinical data presently available demonstrating increased resistance of Carpentier-Edwards bioprostheses to calcification as compared to other commercially available porcine valves. **Caution:** Federal (USA) law restricts this device to sale by or on the order of a physician. Baxter International Inc. is the owner of the following US patent which covers the products described herein: 4,106,129. Patent numbers in foreign countries supplied upon request. See package insert accompanying product for a full discussion of warnings, contraindications, complications and prescribing information. Carpentier-Edwards® is a registered trademark of Baxter International Inc.

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Original Articles

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"Certain thoracic diseases which for generations have fallen to the lot of the surgeon are yet badly handled. Conspicuous among these is that patriarch of the surgical scrapheap, chronic empyema. There is obvious opportunity for improvement in the treatment of this disease." Thus, Samuel Robinson wrote in 1915 [1]. His words are equally appropriate for the 1990s. As shown by Horrigan and Snow in this issue [2], there continues to exist a group of patients with chronic pleural space infection in whom the lung cannot be expanded because of either intrinsic pulmonary disease or prior pulmonary resection. How should these patients be managed?

John Alexander [3] in his classic work on pulmonary tuberculosis demonstrated that thoracic infections often had two components, parenchymal and pleural, both of which had to be dealt with adequately for successful management. Parenchymal infections, which in the past were intractable and often fatal, are now readily cured with a variety of chemotherapeutic agents. At times, however, the involved lung becomes destroyed and develops into a site for chronic infection, which then must be resected before the infection can successfully be brought under control.

See also page 695.

Pleural infection requires obliteration of the pleural space, often in the early stage simply by tube thoracostomy drainage, but as the infection becomes chronic, open drainage may be necessary, especially if signs and symptoms of sepsis are present. Robinson was well aware of these principles [1, 4]. In 1915, he performed open pleural drainage by making a U-shaped skin incision on the chest wall over the empyema. The cavity was entered by removing the overlying ribs and then debrided. Finally, the skin edges were approximated to the empyema cavity. This procedure is remarkably similar to the procedure described by Leo Eloesser 20 years later in 1935 that eventually became known as the "Eloesser flap" [5]. Obviously, Eloesser was not the first to perform the procedure that bears his name.

If the lung does not expand after drainage, further decisions must be made regarding management of the pleural cavity. In some patients who are severely debilitated or who have infections related to incurable cancer, drainage alone may be all that is required to prevent recurrent sepsis. Patients who improve and who have empyema from inflammatory disease, however, are candidates for further operative procedures first to control bronchopleural fistula and then to obliterate the pleural space.

Management of bronchopleural fistula has been variable and, depending on the size, has ranged from simple pleural drainage to thoracoplasty to direct closure. As pointed out by Horrigan and Snow, closure of a large fistula either before or at the time of pleural space obliteration is mandatory for successful management. Like these authors, we prefer direct closure followed by reinforcement with muscle transposition.

Over the years, three different methods of obliterating pleural space have evolved. Historically, the earliest was thoracoplasty to collapse the space. Before 1890, rib resection with resection of intercostal muscle and parietal pleura was performed by Schede, Estlander, and Simon [6]. However, the modern-day version of extrapleural subpericostal thoracoplasty, as applied by John Alexander in the late 1930s to collapse therapy for tuberculosis, established the principles for the safe performance of this procedure [3]. The effectiveness of this procedure depends on the extent to which the pleural cavity can be collapsed. If complete, successful management is common but at the expense of both functional and cosmetic deformity.

The second method of pleural obliteration was to fill the space with autogenous tissue such as lung or muscle. Fowler from England in 1893 [7] and Delorme from France in 1894 [8] were the first to propose decortication as a method of expanding a collapsed lung that had been entrapped by chronic infection. Since their original descriptions, the Fowler-Delorme technique of pulmonary decortication has been modified to remove the entire empyema cavity and throughout the years has been the mainstay of successful pleural space obliteration. The use of muscle flaps in obliterating pleural space is not new. Robinson obliterated these spaces with intrathoracic transposition of latissimus dorsi muscle [1, 4]. Although he successfully treated 6 patients with open pleural drainage and muscle transposition, this technique never became popular until the 1980s [9]. Since then a variety of different muscles and omentum have been transposed into the thorax to control infection with great success and with minimal functional and cosmetic disability.

The last method of managing the pleural space is sterilization of the space with antimicrobial solutions. This procedure was originally performed by Heuer [6] in 1913 when he successfully treated a patient with tuberculous empyema by repeated injections of bismuth paste into the

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cavity. Since that time, tube thoracostomy combined with irrigation of the empyema cavity has been used with varying degrees of success. It was not until 50 years later, however, that further refinement of this technique occurred. In 1963, Clagett and Geraci [10] demonstrated that the treatment of choice for postpneumonectomy empyema was open pleural drainage, and, when the pleura was clean, secondary chest wall closure after first filling the pleural cavity with antibiotic solution. Surgeons who have used the "Clagett procedure" in the treatment of postpneumonectomy empyema have generally been satisfied with the method and have achieved satisfactory results in the majority of their patients [11]. Although sterilization techniques have infrequently been used in the treatment of persistent nonpneumonectomy empyema, this is a valid indication for this technique if there is no evidence of bronchopleural fistula.

Although management of the chronically infected pleural space has changed over the years, the goals of therapy remain the same—to conserve the patient's life with a healed chest wall without evidence of infection. Determination of which techniques are necessary to achieve these goals must be tailored to the individual patient. Obviously, many questions need to be answered. Is the patient otherwise in good health? Is the entrapped lung capable of expansion? Is there an associated bronchopleural fistula? Is local muscle available for transposition into the chest? Ultimately the general thoracic surgeon must carefully balance the ability of each technique to achieve the desired result against the mortality and morbidity associated with that particular technique. Horrigan and Snow have demonstrated that successful management is possible with resection of destroyed lung, closure of bronchopleural fistula, and obliteration of residual pleural space. Using different techniques, including pulmonary resection, muscle transposition, and particularly thoracoplasty, these authors were able to accomplish this in one stage with no operative mortality, complete healing of all wounds, and no late recurrent infections. There was, however, moderate long-term disability in the form of restricted range of shoulder motion and scoliosis.

Clearly, Horrigan and Snow have raised the issue of the role of thoracoplasty in 1990. With the many techniques available today, we believe that extensive thoracoplasty is rarely indicated. Although potentially successful, the functional and cosmetic deformity is great. Does this imply that thoracoplasty should be avoided? We believe not, but when used this procedure should be limited, confining resection to only a portion of a few ribs, and then combined with other space reduction techniques, primarily intrathoracic transposition of extrathoracic skeletal muscle.

We compliment Horrigan and Snow on their excellent results and agree that aggressive attempts at obliteration of the pleural space remain the treatment of choice for most patients with chronic empyema. Our choices differ only in order of preference. We believe that all of these techniques should be a part of the armamentarium of each general thoracic surgeon.

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Behavior of Small Bronchial Carcinomas

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The article in this issue by Ishida and associates [1] covers the spectrum of behavior of lung cancers 3 cm in size or less. We tend to automatically classify these small tumors as T1 lesions, but it is readily apparent from the data presented that a percentage of these lesions, albeit small, are in fact T2 and even T3 or T4 tumors. More importantly, hilar and mediastinal lymph node involvement from these small tumors is more common than one expects. An overall incidence of 29% was recorded and a definite correlation of increasing frequency of lymph node involvement with increasing size of the lesions was evident. Distant metastases were likewise occasionally encountered at the initial diagnosis of these small tumors and were the most common sites of treatment failure in the patients who subsequently died of their disease. This was recorded even in the smallest lesions—less than 1 cm in size—but was most common in the patients whose tumor was 2 to 3 cm in size at the time of initial diagnosis.

The overall long-term survival was related to tumor size but this was not evaluated as to whether or not size alone was an independent variable. The poorer survival in the larger tumors may only reflect the greater frequency of lymph node involvement and distant metastases in this subset of patients with cancers of 3 cm or less in size. However, Read and associates [2] have noted that the size of the tumor per se in patients with T1 N0 M0 cancer is important—they reported that patients with squamous cell tumors smaller than 2 cm had a significantly better survival than those with lesions 2 to 3 cm in size. Similar findings were reported by the Mayo Clinic group in 1969 [3].

See also page 708.

Read and collegues [2, 4] have even suggested that the T1 N0 M0 subset be divided into two groups relative to the lesion's size. The present observations support this view. However, in my opinion, there is little reason to subdivide the T1 descriptor into two categories. The heterogeneity of this subset should be recognized but the subset needs no further subdivision. If this were the case, similar subdivision would be called for in the T2 N0 M0 subset because increasing size of the tumor is also a significant detrimental factor in this latter group [5, 6].

Regardless of the biological explanation for the poorer survival of the patients with the larger of these small lesions—the tumor mass alone, the more frequent local spread, the increased incidence of lymph node metastases and distant metastases—the data presented reinforce the concept that all newly discovered solitary lung lesions in middle-aged and older adults, regardless of their size, should be resected unless unequivocal radiographic or histologic proof of their benignancy is obtained. To watch even the smallest of lesions—a solitary lesion is rarely recognized on standard chest radiographs unless it is at least 7 mm to 1 cm in size—or, even worse, to observe those between 1 and 2 cm in size for further growth before a decision is made in favor of resection is untenable. A substantial risk of the patient eventually dying of cancer is incurred if such a course of inaction is taken.

The recognition of the possibility of lymph node involvement, which increases with the size of these lesions, is important in the operative management of the patients with these small tumors. The data presented make compelling the recommendation of the Lung Cancer Study Group that a complete investigation of all ipsilateral mediastinal lymph node stations at operation is a necessity. A thorough mediastinal lymph node sampling or a systematic mediastinal lymph node dissection should be accomplished in all patients even with the smallest of cancers. When this is done, a correct postsurgical stage can be assigned to each patient. Whether the complete node dissection increases the cure rate or is only of prognostic value remains unanswered by the data of Ishida and associates or any other available data. One can surmise, however, that no patient would be cured if metastatic disease in a mediastinal node remains in the thorax after a resection.

Last, the extremely poor prognosis of the small T2 lesions—the result of invasion of the visceral pleura in all but one of the patients reported—and the similar or even poorer prognosis of N1 disease as compared with N2 disease associated with these small tumors is intriguing. Brewer [7] reported the poor prognosis of any tumor that invaded the visceral pleura, and my colleagues and I [8] noted a similar poor prognosis in the T1 N1 lesions a number of years ago. The explanations of these observations remain to be elucidated.

The use by Ishida and associates of the term *incomplete resection* is unfortunate. A 39% actuarial survival was recorded in their incompletely resected group as it was defined by the authors. Obviously, even though a less than optimal resection according to their criteria was done, complete removal of all gross and microscopic tumor must have been accomplished in a number of these so-called incompletely resected patients. An incomplete resection, as I [9] have noted, should strictly be defined as one in which proven gross or microscopic tumor remains in the thorax after the resection. A truly incomplete resection is accompanied by only a negligible survival

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rate, and the term should not be used in the context of a lesser resection or one without an accompanying mediastinal lymph node resection. A more acceptable term would be a *compromise resection* when a less than an optimal but complete tumor resection has been accomplished and no known tumor remains in the hemithorax.

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Choreoathetosis as a Complication of Cardiopulmonary Bypass

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Neurological complications after cardiac operations involving cardiopulmonary bypass (CPB) have a variety of causes. Most are readily apparent, for example, cerebral embolism or hemorrhage or hypoxia, and the lesion responsible is visible on computed tomographic scanning. In addition, with the exception of severe diffuse hypoxic damage, the distribution of neurological signs varies from one patient to another, and is defined by the vascular territory or parts of the brain involved.

Choreoathetosis, one of these complications after CPB, does not come into any of these classifications. The full-blown syndrome is rare, and it is tempting to think that severe sustained choreoathetosis may be a different entity from transient mild chorea. The cause is unknown and it is not accompanied by consistently detectable abnormalities on computed tomographic scan, electroencephalography, or any other currently available investigation including brain histology, although in one report [1] abnormalities were detected in the basal nuclei, the likely site of the lesion. The clinical picture is stereotyped and striking and cannot go unrecognized, although it is almost certainly underreported. No patient has been reported outside early childhood. The onset is from 2 to 7 days postoperatively and in most cases there is a clear period of normality beforehand. The severity usually progresses over 1 to 3 weeks and recovery is usually complete.

See also page 714.

Most reports of choreoathetosis have implicated profound hypothermia combined with circulatory arrest as the guilty party and, under these circumstances, the arrest temperature has varied between 15° and 20°C, except for 1 patient at 9°C [2–6]. The circulatory arrest times have varied between 7 and 71 minutes. The syndrome is thus not only associated with longer arrest times. Only 1 case of severe sustained choreoathetosis has been encountered in Auckland. Characteristically it appeared on day 3 after an initially smooth postoperative course in a 4-year-old child having his first operation, which was totally uneventful. The arrest temperature was 18°C nasopharyngeal and the arrest time was 25 minutes. Two milder cases were reported by us in 1970 [4]—these children were aged 8 and 17 months.

We are indebted to DeLeon and colleagues [7] for the article in this issue that reports the occurrence of severe choreoathetosis in 8 children aged 1 to 5 years after cardiac operations that employed hypothermic CPB without circulatory arrest. Their report confirms the suspicion gained from the literature of the early 1960s that hypothermic CPB alone can cause choreoathetosis. In these early reports [8, 9], the esophageal temperature varied between 5.8° and 16°C, and it was implied that hypothermia below 16°C was dangerous to brain function. It is somewhat strange that the many others currently using profound hypothermia without circulatory arrest for congenital heart disease operations have not also reported some examples of choreoathetosis, although the recent report from Ferry [10], which involved a survey of six major pediatric centers using deep hypothermia, implies that some units do see it in the absence of circulatory arrest.

DeLeon and colleagues concluded that hypothermic CPB below 25°C rectal predisposes to the occurrence of choreoathetosis when it is associated with a longer cooling time (defined as the length of time the perfusate was kept at or below 25°C) and a higher perfusion flow rate. However, in only one of the affected children was the rectal temperature lowered below 20°C. Most cardiac surgeons believe that hypothermia, at least to a lower limit of 18°C nasopharyngeal, is protective to the brain during CPB. Moreover, a longer cooling time is thought to provide more uniform brain cooling than short cooling times and to be preferable, at least when followed by a period of circulatory arrest. Finally, concern has frequently been expressed that brain damage can follow prolonged periods of low flow and does not occur with high flow per se [11].

Interpretation of the study of DeLeon and associates presents problems in a number of important respects. First, the range of each of the variables in their 8 children considered to be significant is too wide to be credible. Thus the rectal temperature was 16° to 25°C and the CPB time 75 to 270 minutes with a cooling time of 30 to 179 minutes; the flow rate varied between "low flow" and 2,500 mL · m^{-2}. Before accepting any of the conclusions offered as significant, a multivariate logistic regression analysis of the data is imperative. Second, the assessment of the patients lacks detail with regard to possible preoperative central nervous system impairment (how many had adverse perinatal events such as low birth weight for gestational age, small head circumference, developmental delay, or overt neurological findings?), for in some patients these may be related to postoperative neurological

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and developmental status including choreoathetosis [3–5]. In addition, imaging of the brain preoperatively sometimes demonstrates lesions unsuspected clinically that may influence late progress [12]. Clearly the 8 children described belong to a group of severely affected individuals with complex congenital heart disease, and most had a prior intracardiac operation involving hypothermic CPB, which may have been associated with prolonged low flow CPB or particulate embolization, or severe postoperative hypotension with low cardiac output. The suggestion that the hypothermic repair was the factor contributing to choreoathetosis after the reoperation has nothing to support it.

Choreoathetosis can occur in a variety of situations disassociated with cardiac surgery. We have seen 1 infant who had choreoathetosis of unknown cause of similar degree before and after operation. Movement disorders may be seen as a side effect of drug therapy [13].

The truth of the matter is that many hundreds of children have undergone hypothermic CPB at temperatures below 25°C and flow rates at or about 100 $\mathrm{mL} \cdot \mathrm{kg}^{-1} \cdot \mathrm{min}^{-1}$ and "cooling times" greater than 1 hour, with and without circulatory arrest, without experiencing choreoathetosis. What then is the missing link? The choreoathetosis is presumably due to hypoxic damage to the basal nuclei secondary to a disturbance of cerebral blood flow triggered by some aspect of the CPB technique, with or without the addition of circulatory arrest. The acid-base status could be particularly important in view of its influence on cerebral blood flow, and preoperative cerebral damage may make its occurrence more likely. Detailed reporting of all the factors listed, together with the drugs used perioperatively, should be added to the protocol in the hope of solving the riddle.

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Thoracoplasty: Current Application to the Infected Pleural Space

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Thoracoplasty, once commonly used in the management of cavitary pulmonary disease, continues to find application in the obliteration of infected pleural spaces. This study reports a series of 13 patients receiving thoracoplasty between 1976 and 1989. Five patients had chronic apical empyema spaces without prior resection of lung tissue. Two of the empyemas were due to tuberculosis, two were due to atypical mycobacteria, and one was due to postpneumonic empyema. All patients had extensive destruction of upper lobe tissue. Eight patients had undergone prior pulmonary resection; 3 had persistent infected spaces in the early postoperative period, 3 had development of empyemas and bronchopleural fistulas late (5 to 19 years) after pulmonary resection, and 2 had postpneumonectomy empyema. All patients had rigid cavity walls preventing space obliteration by rib removal alone and required concomitant resection of the thickened pleura and intercostal muscle tissues. Bronchopleural fistulas were present in 11 patients and were closed with adjacent nonintercostal muscle. All patients survived and had successful obliteration of the infected spaces with acceptable physiological and cosmetic results. We conclude that thoracoplasty remains a useful procedure in the management of the infected pleural space in select patients.

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It is slightly more than 100 years since European surgeons first applied thoracoplastic procedures to the chronically infected pleural space [1]. Numerous individual techniques evolved, and in the 1930s and 1940s, an entire generation of thoracic surgeons became well versed in the application of thoracoplasty, predominantly for collapse therapy of cavitary pulmonary tuberculosis. Over the past several decades, the use of thoracoplasty has been progressively declining because of improvements in antimicrobial therapy and increasing safety of pulmonary resection. Despite these advances, there remains a small group of patients with chronically infected pleural spaces in whom the lung cannot be expanded because of intrinsic disease or prior resection, either partial or total. Although these patients can be managed by the more conservative procedures of rib resection and open tube drainage [2] or Eloesser flap construction [3], these methods have obvious disadvantages in the otherwise healthy patient. This report details our experience with the application of thoracoplasty to this group of patients.

For editorial comment, see page 689.

Material and Methods

Patient Population

Between 1976 and 1989, 13 patients underwent thoracoplasty procedures at Cleveland Metropolitan General Hospital. This group represents approximately one fifth of all patients with pleural space infection complicated by unexpandable or absent lung tissue. Patients who did not receive thoracoplasty were either medically unsuitable (advanced age, uncontrolled cancer, debility) or refused the procedure. These patients were managed by open tube drainage or Eloesser flap construction. The 13 patients having thoracoplasty are grouped according to whether or not they had undergone prior pulmonary resection.

APICAL EMPYEMAS WITHOUT PRIOR RESECTION. This group of 5 patients was seen with extensive destruction of pulmonary tissue from the disease process, often with destruction of the entire upper lobe (Table 1; Fig 1). The size of the apical space varied considerably, depending on the degree to which the upper lobe had contracted before the development of the subsequent empyema. All patients in this group had bronchopleural fistulas, and the required thoracoplasty size ranged between four and seven ribs. All these patients had chronic fever and weight loss, and 3 had recurrent clinically significant hemoptysis.

POSTRESECTION SPACES. There were 8 patients in this group (Table 2). Six had undergone prior lobectomy, bilobectomy, or resection of giant apical bullae. One patient who had had bilobectomy also had undergone resection of the superior segment of the lower lobe (for coccidioidomycosis). Three patients had persistent fever in the early postoperative period (<3 months), and their spaces failed to resolve with intercostal tube drainage. Apical spaces developed late (5 to 19 years) after partial

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Table 1. Apical Empyema Without Prior Resection

Patient No.	Sex	Age (y)	Organism	Prior Procedure	Ribs Resected
1	M	54	*M tuberculosis* + mixed pyogens	Rib resection + open drainage	1–5
2	M	63	Mixed pyogens	None	1–6 + scapula
3	M	59	*M tuberculosis* + mixed pyogens	Rib resection + open drainage	1–6 + scapula
4	M	64	*M avium intracellulare*	None	1–7 + scapula
5	M	62	*M avium intracellulare* + *A niger*	None	1–4

A = *Aspergillus;* M = *Mycobacterium.*

lung resection in 3 patients; the residual lung had failed to expand completely in the early postoperative period, leaving a fluid cap in the apex of the pleural cavity which later became infected, with the development of bronchopleural fistulas in each (Fig 2). Two patients with postpneumonectomy empyema underwent thoracoplasty: 1 patient had a bronchopleural fistula, and the other had a failed topical antibiotic sterilization of the infected pleural space, or Clagett procedure [4].

Operative Technique

All procedures were performed in one stage. Subperiosteal rib resection was performed first, exposing the rigid, thickened underlying pleural membrane. All empyema cavities were found to be so rigid that rib resection alone failed to provide adequate collapse of the cavities. The empyema cavity was entered, and the intercostal muscles, neurovascular bundles, and thickened empyema cavity wall were resected, with liberal use of electrocautery. It is important to resect enough of the cavity wall so that a residual space is not permitted by a shelving edge or overhanging lip. The serratus posterior superior and rhomboid muscles were preserved and used for fistula closure when indicated. The first rib was resected in all patients, as were the transverse processes of the appropriate vertebrae.

Once the empyema cavity wall had been resected, the exposed lung tissue or mediastinum was inspected for bronchopleural fistulas; they were present in 11 patients and were small and multiple in 9. The fistulas were occluded with small slips of viable muscle sutured into the openings with fine silk. Adjacent trapezius, latissimus, serratus, or rhomboid muscle was sutured to the raw apical lung tissue over the fistula repair to reinforce the fistula closure. The shoulder was mobilized to evaluate whether or not the scapula tended to lock on the uppermost residual rib; if it did, the lower third of the scapula was also resected. The extent of the thoracoplasties were as follows: four ribs in 4 patients, five ribs in 2 patients, 6 ribs in 4 patients, and seven ribs in 3 patients.

Results

All patients survived operation and were discharged from the hospital. Two patients required revision for inadequate space obliteration based on postoperative chest roentgenograms. In 1 patient, the first rib had been intentionally left, and its removal provided satisfactory collapse of the cavity. The other patient was reoperated on to remove an additional lower rib. A third patient with extensive atypical tuberculous infection of the residual lung required tracheostomy and prolonged ventilatory support but was ultimately discharged. The thoracic drains were removed from 6 patients during the hospital admission; in the remainder, small air leaks persisted at the time of hospital discharge, and the large drain was exchanged for a small-caliber rubber tube after discharge; this was removed 2 to 6 months later.

In the late postoperative period, all patients had permanent, satisfactory control of the infectious process with no late recurrences. One patient died 3 years postoperatively of a myocardial infarction, and 1 patient died at 2 years of carcinoma of the contralateral lung. One patient underwent successful myocardial revascularization 7 years postoperatively.

Comment

Thoracoplasty gained widespread acceptance for the management of cavitary pulmonary disease and chronic pleural space infections in an era noted for its poor antimicrobial therapy and lack of sophisticated ventilatory support. In that period, properly performed thoracoplasty gave results superior to other forms of collapse therapy [1]. The disadvantages of this procedure have been reported to include progressive scoliosis, chronic postoperative pain and hypesthesia of the chest wall, progressive pulmonary insufficiency, and a "mutilating" cosmetic appearance [5, 6]. Although the number of patients presented here is small, we have been favorably impressed with the infrequency of these side effects.

Lindskog [7] intimated that leaving the first rib in place may minimize scoliosis, and in a recent series, Hopkins and associates [8] at Duke left the first rib in 20% of their patients. Gregoire and co-workers [9], reporting a series of thoracoplasties for postpneumonic empyema, were able to achieve adequate collapse by leaving the first rib in all patients. Loynes [10], however, reported that the degree of scoliosis was related to the total number of ribs resected as well as the removal of the transverse processes, rather than to the presence or absence of the first rib. We have been disappointed in the adequacy of collapse when the first rib has been left and advocate its removal. One of our patients required reoperation to resect the first rib to achieve adequate cavity obliteration.

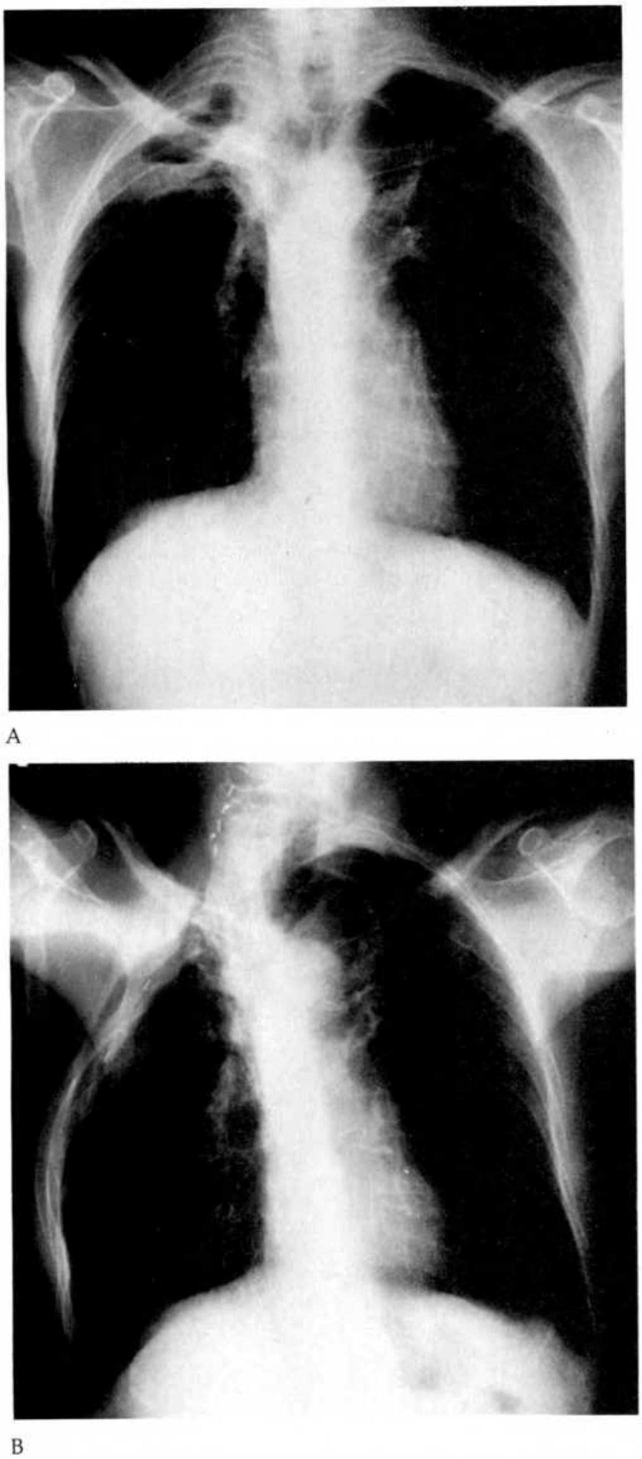

Fig 1. (Patient 5.) (A) Preoperative chest roentgenogram. The right upper lobe has been destroyed by atypical mycobacterial infection, with resultant empyema and bronchopleural fistulas. (B) Chest roentgenogram showing resolution of infected space 8 months after four-rib thoracoplasty.

Table 2. Empyema After Pulmonary Resection

Patient No.	Sex	Age (y)	Prior Resection	Ribs Removed
Early Space (<3 Months)				
6	F	43	RUL, RML, SS (coccidioidomycosis)	1–4
7	M	20	RUL (trauma)	1–4
8	M	68	RUL (cancer)	1–4
Late Space (5 to 19 Years)[a]				
9	F	44	Bullectomy	1–6 + scapula
10	M	64	RUL (tuberculosis)	1–6 + scapula
11	M	49	RUL (cancer)	1–5 + scapula
Postpneumonectomy Empyema				
12	M	67	Pneumonectomy (cancer)	1–7 + scapula
13	F	25	Pneumonectomy (lung abscess)	1–7 + scapula

All of these patients had rib resection and open drainage before thoracoplasty.

RML = right middle lobectomy; RUL = right upper lobectomy; SS = superior segment, lower lobe.

All of our patients have scoliosis to some degree. In none, however, has it been severe, and none have had symptoms referable to the curvature. The cosmetic result has not been unacceptable (Fig 3).

With respect to chronic chest wall pain, only 1 of our patients (patient 10) had long-term incapacitating pain. He was managed by neurosurgical referral and dorsal rhizotomy and achieved acceptable pain relief.

The most troublesome complaint experienced by our patients has been restriction of shoulder motion on the involved side. The scapula becomes adherent to the chest wall, especially if a portion of that bone has been resected, and the appearance is that of a "frozen" shoulder. Abduction and elevation are accomplished by tilting the torso away from the involved side, and this disability has been reasonably well accepted.

We did not see symptoms of progressive pulmonary failure in our patients, as described by Gaensler and Strieder [11] and by Huang and Lyons [12]. These observers noted this finding only in postpneumonectomy patients, and our series has only 2 patients in this category, probably too small a number to be meaningful.

More than half of our patients were discharged with the thoracic drains still in place because of persistent small air leaks. We exchanged the large-bore tubes placed at operation for smaller rubber catheters after several weeks. In the first years of this series, these bronchocutaneous fistulas were evaluated by periodic sinograms, and the tubes were removed only when there was no evidence of a bronchial communication. More recently, believing that a new space would not develop in the midst of a firmly healed wound, we have simply waited several months

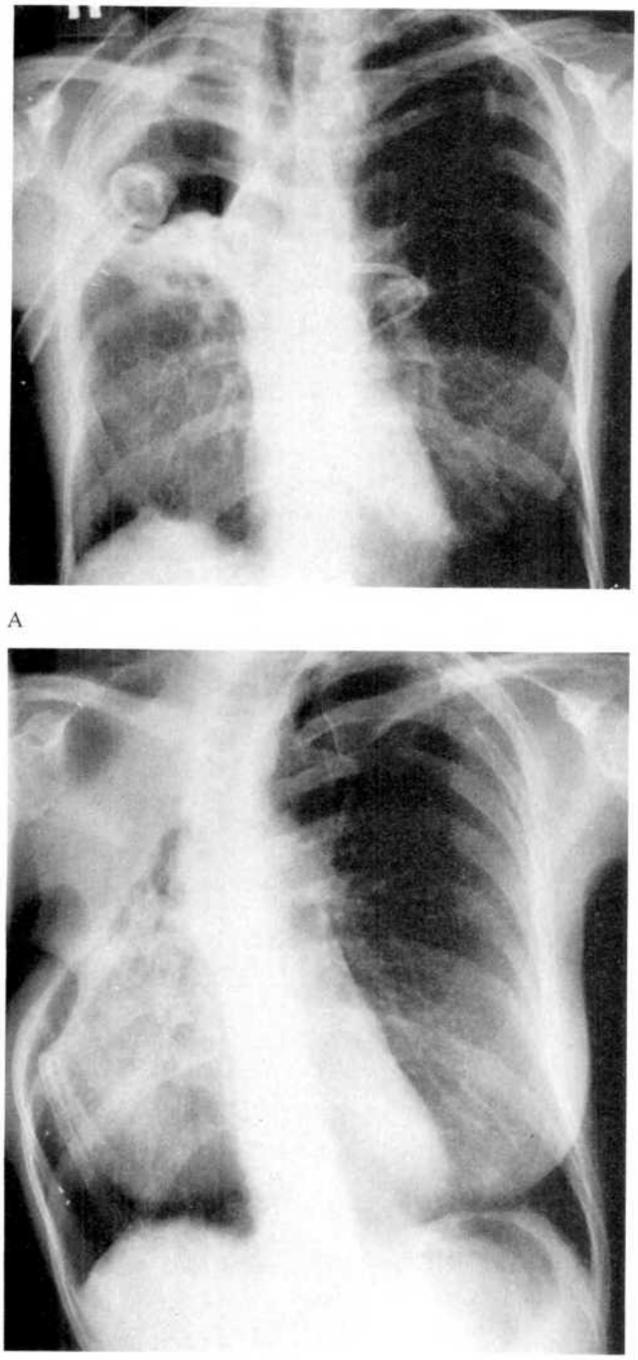

Fig 2. (Patient 9.) (A) Preoperative chest roentgenogram. The right lung had incompletely expanded after resection of large apical bullae; 5 years later the apical fluid became infected, and a bronchopleural fistula developed. The space was initially treated by rib resection and open drainage. (B) Chest roentgenogram made 1 year after six-rib thoracoplasty and scapular resection.

until the thoracoplasty wound was securely healed and then removed the catheters. None of the patients displayed a residual space. In contrast to Gregoire and co-authors [9], we believe that meticulous closure of every identifiable pulmonary fistula by "plugging" it with a slip of skeletal muscle is highly effective in reducing postoperative air leaks; this also contributed to earlier tube removal in the patients operated on more recently. As all of our patients demonstrated empyema cavities with thick, unyielding walls, we thought it necessary to resect the cavity walls to effectively obliterate the space. This technique of rib resection combined with excision of the wall of the empyema cavity is generally referred to as a Schede thoracoplasty [2, 5, 6]. The procedure as originally described entailed leaving the wound open, using packing to fill the space, rather than primary wound closure, as we have practiced. It also involved a much more extensive resection of ribs, both in number and extent, than we have done. By confining the rib removal below the third rib to the more posterior aspect of the chest, the severe deformity that characterizes most descriptions of the Schede thoracoplasty can be avoided. Both Wangensteen [13] and Kergin [5] attempted to improve the cosmetic result by leaving the intercostal muscle bundles in place. In our patients, we found the intercostal tissues to have an unreliable vascular supply after rib resection and think that their removal did not detract from the final appearance or function.

Many of the patients in our series had the infected pleural space drained before thoracoplasty by rib resection and open drainage tubes; this group has had the

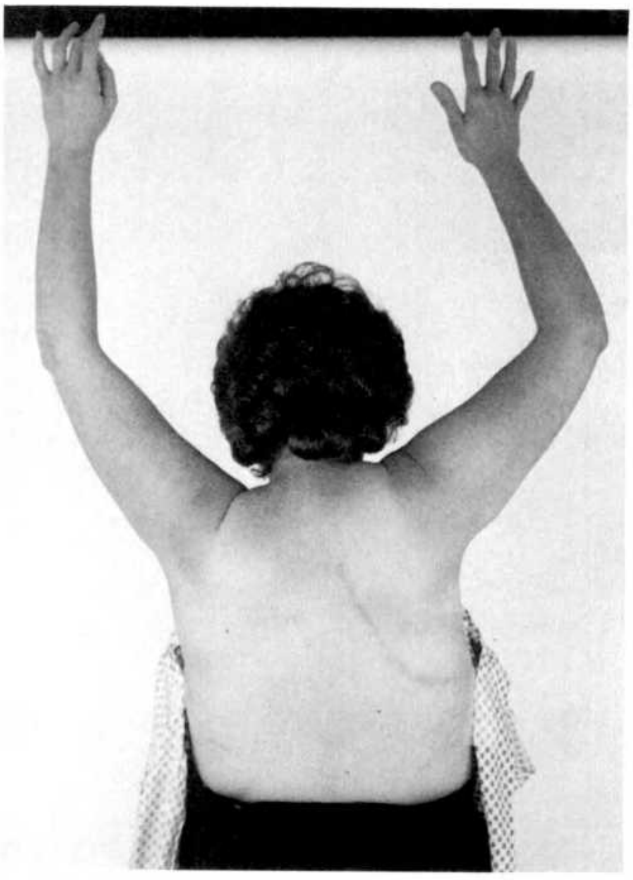

Fig 3. (Patient 13.) Cosmetic result 12 years after seven-rib thoracoplasty and scapular resection for postpneumonectomy empyema.

opportunity to make a comparison of the quality of their lives between long-term drainage and thoracoplasty and has uniformly expressed preference for thoracoplasty as a long-term solution to the infected space. We believe that there remains a useful role for the application of this time-honored procedure in the management of pleural space infection in properly selected patients.

We gratefully acknowledge the contributions to us on this subject by our surgical mentors, Drs Harvey J. Mendelsohn, Jay L. Ankeney, and John F. Perry, Jr.

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DISCUSSION

DR JOHN R. HANKINS (Baltimore, MD): I enjoyed this paper very much and have just one comment. Horrigan and Snow mentioned the transfer of small slips of muscle, but their emphasis seemed to be more on the removal of ribs and much less on the use of chest wall muscles to obliterate the infected pleural space. In the late 1970s, our group at the University of Maryland presented to this Association our experience with the use of chest wall muscle flaps to close bronchopleural fistulas. We found that if we used the larger muscles, such as the pectoralis major or the latissimus dorsi, they helped fill the infected space in addition to closing the fistulas, and we did not have to remove as many ribs. Would the authors please comment on this point?

DR KAMAL A. MANSOUR (Atlanta, GA): I challenge John Alexander's statement. We have now another operation that can close a space. I believe no presentation on management of the infected pleural space is complete without a discussion of muscle flaps. In our institution, my colleagues and I no longer do total thoracoplasties, whether for apical empyemas, postresection spaces, or postpneumonectomy empyemas, nor do we resect the first rib or the scapula anymore. Use of the serratus anterior or latissimus dorsi muscle in connection with a very limited thoracoplasty has obviated the need for total thoracoplasty.

DR THOMAS B. FERGUSON (St. Louis, MO): I have two comments. First, I think the idea of trying to save the chest wall by filling the space with tissue is a good idea. Second, the problem with muscle flaps is that they are not always available, as many of these patients have had previous procedures. I have found that omentum works very well, particularly if the patient has a persistent bronchopleural fistula that you cannot close by other means.

DR SNOW: I think both Dr Hankins and Dr Mansour are absolutely right in terms of trying to fill these spaces with viable tissue if it is available. I was going to make the same comment that Dr Ferguson did, ie, that some of the patients have had a previous thoracotomy, which usually obviates the use of the latissimus, which is a primary large-bulk muscle for these procedures. I agree that to the extent that you can fill the space and save some of the chest wall, it is certainly the best thing to do, if possible.

Repair of Supravalvar Aortic Stenosis: Cardiovascular Morphometric and Hemodynamic Results

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Supravalvar aortic stenosis is associated with normal systolic pressure in the aorta and its branches with the singular exception of the coronary arteries, which are hypertensive. This uncommon lesion has been treated by patch aortoplasty of several types. We examined hemodynamics and morphometrics in 13 patients who underwent operation for supravalvar aortic stenosis from 1979 through 1988. They ranged in age from 2 to 43 years (mean age, 14.5 ± 3.8 years [± standard error of the mean]). There were no operative deaths. Preoperative and postoperative (1 to 5 years) catheterization or echocardiography or a combination of these was done in all but 3 patients (1 died late suddenly without a postmortem examination; 1 was lost to follow-up; and 1 has not yet been restudied). Pressure gradients across the stenosis in patients treated with a single-sinus patch (n = 4) were 65 ± 18 mm Hg preoperatively and 5 ± 3 mm Hg postoperatively ($p < 0.05$) and in patients with a bisinus patch (n = 6), 83 ± 15 mm Hg preoperatively and 6 ± 2 mm Hg postoperatively ($p < 0.05$); the two groups were not significantly different. Measurements of the diameters of the coronary arteries, aortic annulus, and descending aorta were made, and calculation of the ratio of the coronary artery and annulus diameters to the descending aortic diameters both preoperatively and postoperatively was possible in 5 patients. The left coronary artery was larger than normal before and after operation. Preoperatively there was a disproportionate increase in left coronary artery and annulus size during systole. Left ventricular wall thickness decreased significantly postoperatively ($p < 0.05$). Repair of supravalvar aortic stenosis (localized and diffuse) by both single sinus and bisinus patch repair is safe and hemodynamic results are good. The left coronary artery remained enlarged after complete relief of systolic hypertension and regression of left ventricular hypertrophy.

(Ann Thorac Surg 1990;50:700–7)

Supravalvar aortic stenosis is a relatively rare cause (8%) of congenital left ventricular outflow tract obstruction [1]. As in all such obstructions, the left ventricle is hypertensive. However, this lesion is unique because the coronary arteries alone among systemic vessels are exposed to high systolic pressure, which leads to dilatation, tortuosity, and premature atherosclerosis of these arteries [2]. These changes can make patients susceptible to sudden death both before and after repair [3, 4].

Operative repair of the malformation can be accomplished by enlarging the stenotic zone of the aorta with a patch into one sinus of Valsalva or by extending the aortoplasty into two sinuses. Postoperatively, the coronary arteries are exposed to normal aortic pressure; their morphological response to this change has never been documented. We sought to evaluate both the coronary artery morphology after repair and the effectiveness of the two types of repair by retrospective review of our patients.

Material and Methods

We reviewed the medical records of all patients undergoing operation for supravalvar aortic stenosis at the Medical University of South Carolina from 1979 through 1988. Data collected included date of birth, date of operation, sex, height, weight, associated anomalies, medical history, presenting symptoms, chest roentgenograms, and electrocardiograms.

In most patients, echocardiography was done preoperatively, immediately postoperatively, and at varying intervals thereafter. It documented the location of stenosis, the instantaneous systolic pressure gradient across the stenosis (hereafter referred to as aortic gradient), and the presence and severity of aortic insufficiency in patients for whom continuous-wave Doppler echocardiography and color-flow Doppler echocardiography were available. Coronary artery size was measured with high-resolution echocardiography in a few patients, but these data were excluded from use in calculating coronary artery size because of inability to measure systolic and diastolic diameters separately.

Electrocardiograms were evaluated for evidence of ar-

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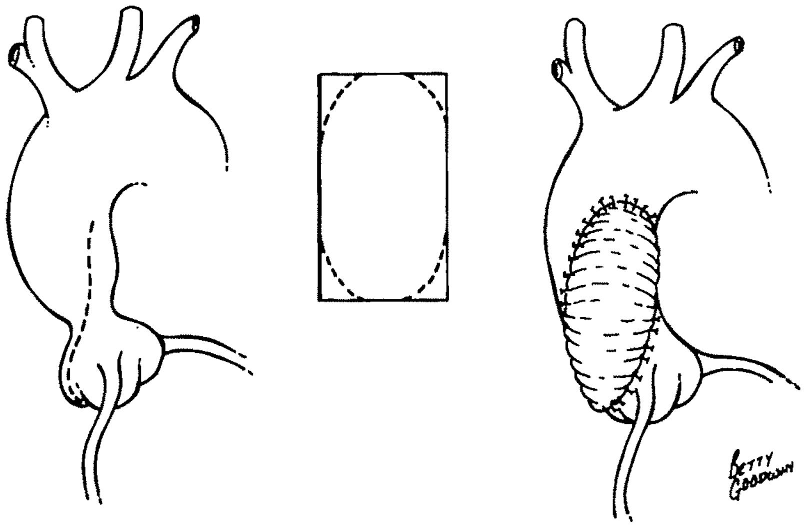

Fig 1. Single-sinus patch technique. After placement of the aortic cross-clamp as close to the innominate artery as possible and administration of cardioplegia, an oblique incision is made across the narrow area from a point well above the stenosis, with great care taken to avoid injuring the aortic leaflets and commissures. The incision is carried into the noncoronary sinus of Valsalva to within a few millimeters of the leaflet attachment to the sinus wall. A patch (our preference is autologous or bovine pericardium because of superior hemostasis) is cut in an oval shape wide enough to enlarge the narrowest area of aorta to the same diameter as the distal ascending aorta. The patch is sutured in place with a continuous nonabsorbable monofilament suture.

rhythmias and left ventricular hypertrophy preoperatively and postoperatively.

Information recorded from cardiac catheterization included peak-to-peak aortic gradient, presence and severity of aortic insufficiency, and associated cardiac lesions. The operation performed, complications, and deaths were recorded.

Morphometric Measurements

All angiographic measurements were performed using selected frames from aortograms or left ventriculograms in the left anterior oblique projection. Diameters of the proximal right coronary artery (RCA), proximal left main coronary artery (LCA), aortic annulus (AAn), and descending thoracic aorta (DAo) near the level of the diaphragm were measured in both systolic and diastolic frames. Left ventricular free wall thickness (LVWT) was measured during diastole in the left anterior oblique projection at the level of the apical border of the mitral valve orifice. These measurements were used to derive relative size values: ratio of RCA, LCA, AAn, and LVWT to DAo. Normal values for RCA/DAo and LCA/DAo were derived from published age-related normal values for RCA, LCA, and DAo size (Appendix 1) [5–7]. The degree of change from normal size of the coronary arteries was evaluated by deriving the ratio of LCA/DAo and RCA/DAo to respective normal values for age. Throughout this report, the terms *systole* and *diastole* refer only to ventricular systole and diastole.

Surgical Technique

All procedures included standard techniques of cardiopulmonary bypass under moderate hypothermia with cardioplegic hypothermic myocardial preservation. All operations were done by 2 surgeons (R.M.S. and F.A.C.), and technique was selected by surgeon's preference. The two methods of repair were single sinus aortoplasty (Fig 1) and bisinus or extended aortoplasty (Fig 2). The method of repair of the diffuse lesion is illustrated in

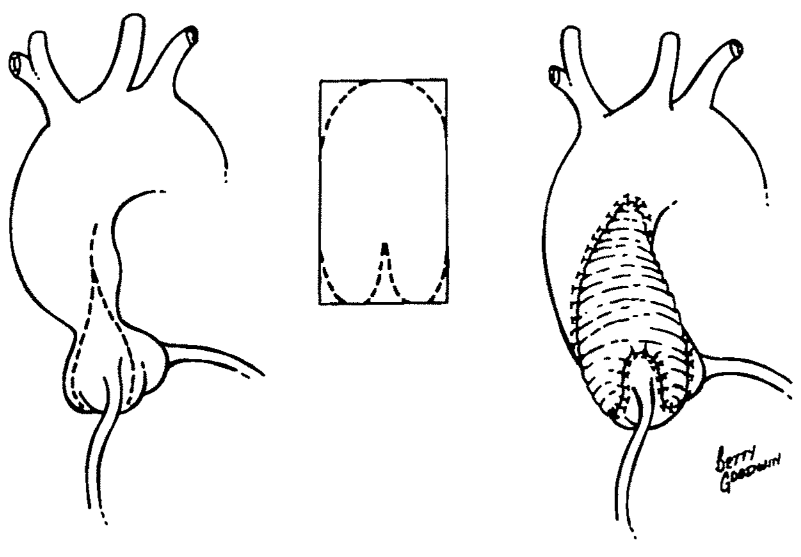

Fig 2. Bisinus patch technique. The technique is the same as for the single-sinus patch, but after the aortotomy is complete, another incision is made into the right coronary sinus a few millimeters from the coronary artery orifice. It also extends to within a few millimeters of the leaflet attachment. A patch is fashioned similar to the single-sinus patch, but a notch is cut from one edge to accommodate the commissure. The suture line is begun at the apex of the commissure and run in both directions with nonabsorbable monofilament suture.

Fig 3. Repair of diffuse supravalvar aortic stenosis. The technique is the same as for bisinus repair, but the repair is begun under deep hypothermic circulatory arrest. Suturing of the patch is started in the descending aorta, and circulation is resumed when the suture lines reach the ascending aorta so an aortic cross-clamp can be placed. The proximal notched edge of the patch is fashioned from excess patch length when the original suture line reaches the proximal ascending aorta.

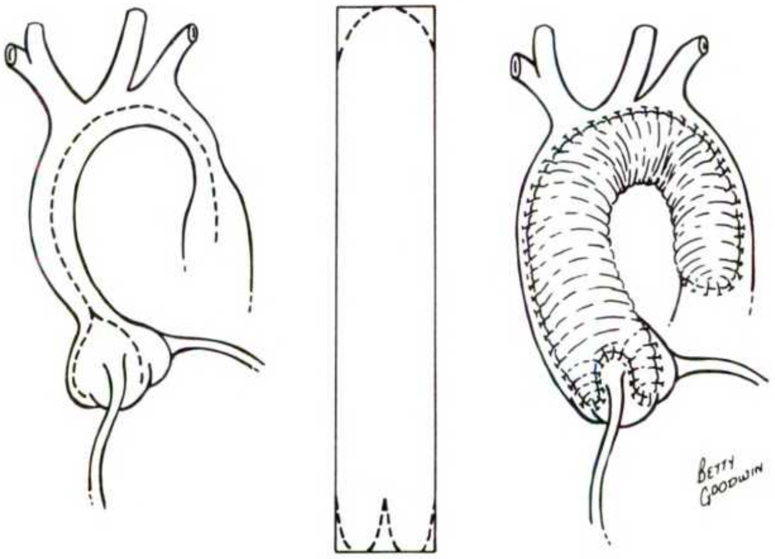

Figure 3. Patch material was glutaraldehyde-fixed autogenous or bovine pericardium, Gore-Tex, or Dacron.

Statistical Analysis

The Wilcoxon signed-rank test was used to compare preoperative with postoperative aortic gradients; preoperative with postoperative and systolic with diastolic LCA/DAo, RCA/DAo, and AAn/DAo; and preoperative with postoperative diastolic LVWT/DAo. The paired *t* test was used to compare LCA/DAo and RCA/DAo with normal values for age and to compare the change from normal of the LCA/DAo with that of the RCA/DAo both preoperatively and postoperatively.

Results

Demographic Characteristics

Thirteen patients were operated on for supravalvar aortic stenosis during the study period (Table 1). The 11 male and 2 female patients ranged in age from 2 to 43 years (mean age, 15 ± 4 years [± standard error of the mean]). Associated cardiac lesions were present in 4 patients

Table 1. Summary of Patient Data

Patient No.	Sex	Age (y)	Morphology	Associated Lesions	Patch Type	Aortic Insufficiency		Aortic Gradient (mm Hg)	
						Preop	Postop	Preop	Postop
1	Male	2	Local	Subaortic stenosis + VSD	Bisinus	None	. . .	25	Not yet studied
2	Female	6	Local	None	Bisinus	None	Mild	56	7
3	Female	8	Local	None	Single sinus	None	Mild	100	0
4	Male	8	Local	None	Single sinus	None	None	90	7
5	Male	8	Diffuse	None	Bisinus	None	None	100	0
6	Male	9	Local	None	Single sinus	None	Mild	45	12
7	Male	9	Local	None	Single sinus	Mild	Mild to moderate	25	0
8	Male	11	Local	None	Bisinus	None	Trace	45	10
9	Male	13	Diffuse	None	Bisinus	None	None	120	0
10	Male	19	Local	Ventricular aneurysm	Single sinus	Mild	. . .	65	Lost to follow-up
11	Male	30	Local	ASD	Bisinus	None	None	128	15
12	Male	30	Local	Mitral insufficiency	Bisinus	None	. . .	75	Dead
13	Male	43	Local	None	Bisinus	None	None	51	5

ASD = atrial septal defect; VSD = ventricular septal defect.

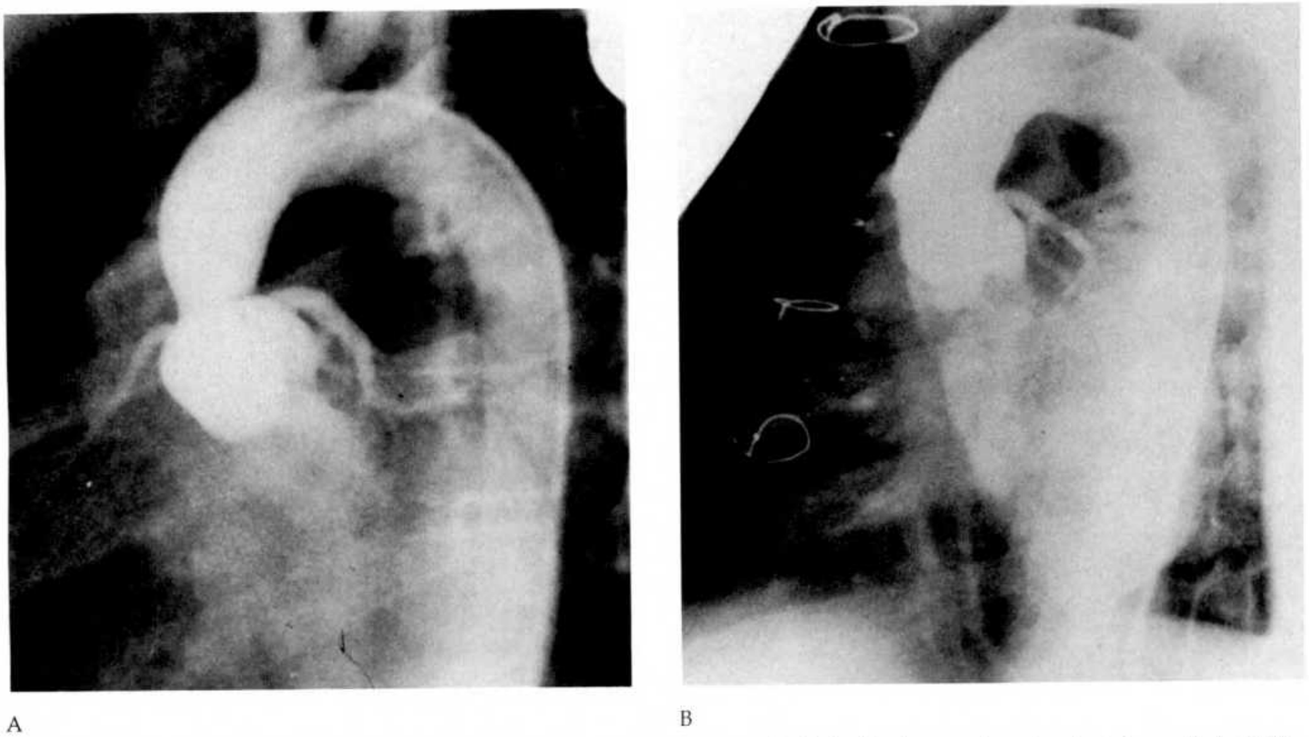

Fig 4. (A) Preoperative left anterior oblique projection of aortogram in patient 3, a girl with localized supravalvar stenosis and a gradient of 100 mm Hg. (B) After single-sinus patch repair, left anterior oblique projection of aortogram shows wide open supravalvar area. No gradient was found at this cardiac catheterization.

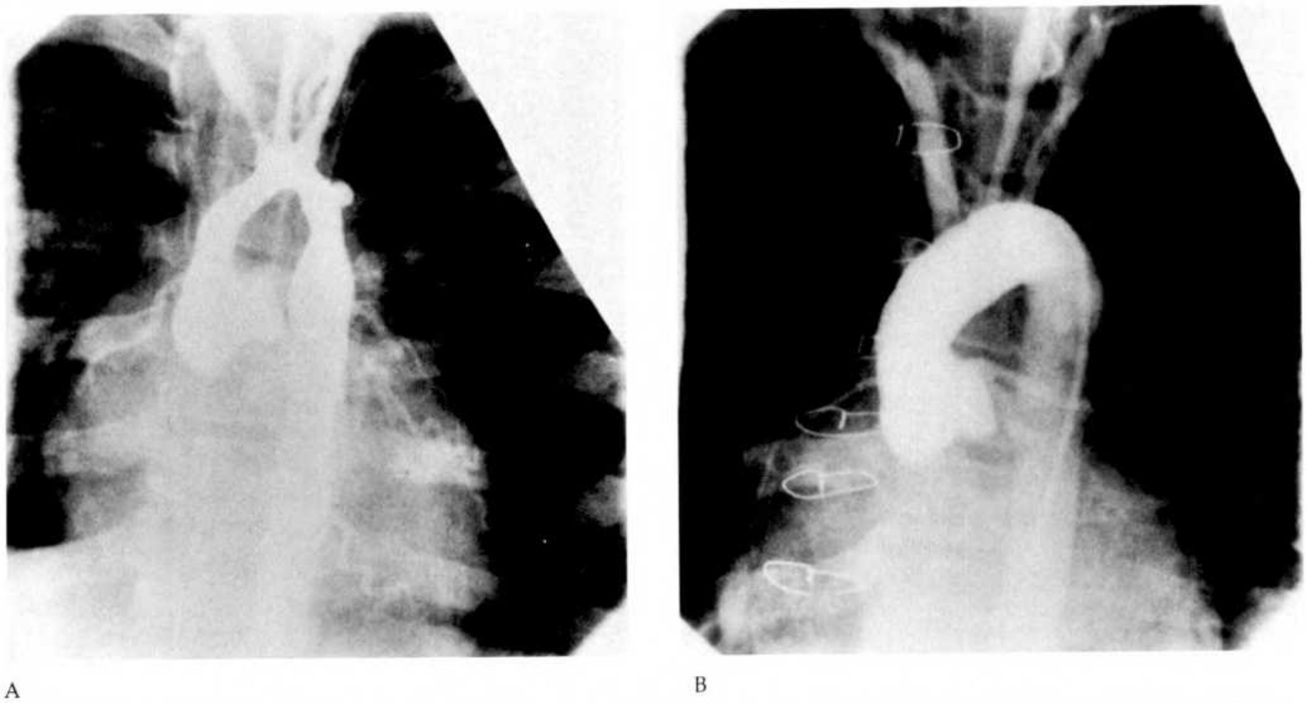

Fig 5. (A) Preoperative left ventricular projection of aortogram in a boy with Williams syndrome (patient 9), who had a gradient from the left ventricle to the descending aorta of 120 mm Hg and diffuse stenosis extending into the descending aorta. There were two small saccular aneurysms of the aortic arch, one of which is seen here, and stenosis of the origins of the arch branches. (B) After bisinus patch repair with excision of the aneurysms and Gore-Tex graft bypass of the innominate origin, there was no residual gradient and the appearance of the reconstructed aorta was excellent.

Table 2. Comparison of Preoperative and Postoperative Aortic Gradients

Group	Preop (mm Hg)	Postop (mm Hg)
Entire (n = 10)	76.0	5.6[a]
Bisinus repair (n = 6)	83.3	6.2[a]
Single-sinus repair (n = 4)	65.0	4.8[a]

[a] Significance: $p < 0.05$ between the preoperative and postoperative values.

(mitral insufficiency, left ventricular aneurysm, atrial septal defect, and subaortic stenosis with ventricular septal defect). Seven patients had evidence of Williams syndrome (elfin facies, hypercalcemia, and mental retardation) [8]. One patient, 2 years old at the time of bisinus repair, had undergone repair of a ventricular septal defect and subaortic stenosis at 3 days of age. It is not certain whether the supravalvar aortic stenosis was congenital or caused by stenosis at the closure site of the original aortotomy through which subaortic tissue was excised [9].

Eight (62%) of the 13 patients were symptomatic with fatigue (4), syncope (2), chest pain (1), or congestive heart failure (1). The chest roentgenogram was interpreted as normal in 9 patients, and cardiomegaly was the only abnormality noted in 4. Electrocardiography was normal in 4 patients, 8 had evidence of left ventricular hypertrophy, and 1 had atrial fibrillation with interventricular conduction defect (this patient also had mitral insufficiency).

Angiographically, the area of stenosis was localized to the ascending aorta in 11 patients (Fig 4) and extended beyond the aortic arch in 2 (Fig 5), brothers with familial Williams syndrome.

Surgical Results

In addition to repair of the supravalvar obstruction, 3 patients had concomitant operations: closure of an atrial septal defect, resection of a left ventricular aneurysm, and mitral valve replacement. One patient had undergone previous repair of a ventricular septal defect and subaortic stenosis, as already noted.

There were no operative deaths. The single late death occurred in the patient with concomitant mitral valve replacement, who died suddenly at home several weeks after hospital discharge. No postmortem examination was done.

Postoperative complications included a left phrenic nerve palsy in a patient whose repair included aortic arch reconstruction. In another patient, pericardial effusion caused by postpericardiotomy syndrome was treated by pericardial window.

One patient was lost to follow-up before any postoperative evaluation could be done, and 1 is awaiting postoperative study. Therefore, postoperative data were available on 10 patients.

Hemodynamic Measurements

Preoperative echocardiography was done in 12 of the 13 patients, and in 7, aortic gradients were measured by continuous-wave Doppler echocardiography. Postoperative echocardiography was performed in 9 patients from 11 to 63 months postoperatively (mean, 34 months).

All patients underwent cardiac catheterization preoperatively and 5, 1 year to 5 years (mean interval, 33 months) after operation (see Figs 4, 5). The decrease in aortic gradient from the preoperative to the postoperative measurement (n = 10) was significant (Table 2). There was no difference between single-sinus and bisinus repair.

Preoperatively, 2 patients had mild aortic insufficiency. After operation, aortic insufficiency persisted in 1 of these patients (single-sinus repair), and the other was lost to follow-up. New (trace to mild) aortic insufficiency appeared in 4 (44%) of the other 9 patients, 2 with single-sinus and 2 with bisinus repair (not significant). Among the 5 patients with aortic insufficiency immediately postoperatively, serial follow-up echocardiography 11 to 63 months after operation (40 ± 11 months) showed no change in degree of insufficiency.

Morphometric Evaluation

Among the 5 patients with both preoperative and postoperative cardiac catheterization, the LCA/DAo and the AAn/DAo preoperatively were significantly higher during systole than during diastole (Table 3). There were no other significant differences between systolic and diastolic or between preoperative and postoperative sizes.

When normalized for age, the LCA/DAo was larger than the RCA/DAo preoperatively ($p < 0.05$) but not postoperatively (Table 4). The LCA/DAo in systole was larger than normal for age preoperatively ($p = 0.05$) and postoperatively ($p = 0.08$).

The LVWT/DAo decreased from 1.31 ± 0.29 preoperatively to 0.96 ± 0.11 postoperatively ($p < 0.05$). The left ventricular hypertrophy on electrocardiography that was

Table 3. Comparison of Coronary Artery Size and Annulus Size in Systole and Diastole

Time	RCA/DAo		LCA/DAo		AAn/DAo	
	Systole	Diastole	Systole	Diastole	Systole	Diastole
Preop	0.30 ± 0.09	0.23 ± 0.03	0.43 ± 0.08	0.32 ± 0.05[a]	1.96 ± 0.16	1.72 ± 0.13[a]
Postop	0.25 ± 0.06	0.26 ± 0.04	0.35 ± 0.05	0.31 ± 0.02	1.66 ± 0.04	1.68 ± 0.10

[a] Significance: $p < 0.05$ versus systole.

AAn = aortic annulus; DAo = descending thoracic aorta; LCA = left coronary artery; RCA = right coronary artery.

Table 4. Coronary Artery Size Normalized for Age

Time	RCA/DAo	LCA/DAo
Preop	1.51 ± 0.44	1.93 ± 0.35[a]
Postop	1.35 ± 0.37	1.53 ± 0.24

[a] Significance: $p < 0.05$ versus RCA/DAo.

DAo = descending thoracic aorta; LCA = left coronary artery; RCA = right coronary artery.

present preoperatively in these 5 patients became normal in 3, was reduced in 1, and was unchanged in 1.

Comment

Supravalvar aortic stenosis was first repaired successfully in 1961 by patch enlargement of the noncoronary sinus of Valsalva [10, 11]. Later techniques included resection of the stenosis with end-to-end anastomosis [12], primary excision of the obstructive end-aortic ridge, and left ventricular apicoaortic conduit to bypass the lesion [13].

In 1977, Doty recommended bisinus repair by extending a bilobed patch into both the noncoronary sinus and the right coronary sinus for a wider and anatomically more symmetrical repair [4]. This technique is designed to reduce the incidence of postoperative aortic gradients and insufficiency as well as to avoid fibrous proliferative obstruction of the sinuses of Valsalva and the coronary ostia [14–16]. Bisinus and single-sinus patch angioplasty with or without resection of the intimal ridge are currently the most widely used methods of repair.

In our series, 8 patients had bisinus repair and 5 had single-sinus patch repair. There were no operative deaths in either group.

Hemodynamics

Patch angioplasty has been shown to be quite effective in reducing the supravalvar gradient, but there have been reports of a persistent gradient across the region of the aortic root [17]. Data from several reports [14, 18–21] demonstrate the postrepair gradient to average 15 to 30 mm Hg. The attachment of the right and left coronary cusps to the fibrous intimal ridge after patch enlargement of the noncoronary sinus may account for some of these postrepair aortic gradients [17, 20].

In our series, the postoperative gradient was extremely low in both groups, the highest single gradient being 15 mm Hg. The absence of important gradients in our patients may have been due to the use of a large patch, extending from the valve hinges in the sinuses of Valsalva to the ascending aorta well above the obstructive waist and wide enough to produce a normal aortic diameter at the level of obstruction.

A special problem is presented by patients with the diffuse form of supravalvar aortic stenosis. Because relief of the pressure gradient by patch repair is often incomplete [14, 17], a left ventricular–aortic conduit has been recommended [18]. Our 2 patients with this form of the disease (patients 5 and 9) had excellent results with bisinus patching and extension of the patch into the descending aorta.

Aortic insufficiency after repair has been attributed to persistent attachment of the right and left coronary leaflets to the residual fibrous intimal ridge [17, 20]. Although more of our patients were free from new aortic insufficiency after bisinus (4/6) than after single-sinus repair (1/3), the difference was not significant, so firm comparative conclusions are not possible. The overall incidence of new aortic insufficiency (44%) in this study was higher than in other reports (range, 0% to 34% [17, 19, 20]), but the lesion was trivial or mild in nearly every instance. The reason for the increased incidence is not clear. It may be related to the use of a large patch that provided good relief of obstruction but contributed to the development of valve insufficiency. We believe it more plausible, however, that the increased incidence is related to highly sensitive techniques now available for detecting mild aortic insufficiency, such as color-flow Doppler echocardiography. In an earlier era, the trivial to mild abnormalities we found may have been undetected. Our follow-up is too short to evaluate stability or progression of insufficiency.

Thus, with respect to the operative mortality and morbidity and the postoperative hemodynamic result, neither method of repair in our series had an advantage over the other.

Morphometrics

The coronary arteries in supravalvar aortic stenosis become dilated and tortuous with evidence of medial degeneration and premature atherosclerosis [3]. Atherosclerotic changes in the coronary arteries and left ventricular hypertrophy caused by outflow tract obstruction have been postulated to be the causes of sudden death both before and after operation. Previously, changes in the size of coronary arteries and in LVWT after repair have not been documented.

We measured relative size of the coronary arteries and AAn by deriving their ratio to the diameter of the DAo. Our reasoning was that this ratio should not change from systole to diastole if compliance of the vascular structures was similar, unless distending pressures were different. This rationale seems to have been correct because the LCA and aortic annulus ratios were significantly higher in systole than in diastole before repair, when aortic root pressure was still high. After repair, when the aortic root pressure was not elevated, the ratios were the same in systole and diastole. The same observations were made in regard to the RCA, but the systolic change was not significant.

We found that LCA size during systole before operation was significantly larger than normal for age and larger than the RCA. This difference in size may be accounted for by selective left ventricular hypertrophy. After operation, the LCA was significantly smaller in systolic size but not in diastolic size. This suggests that postoperative decrease in coronary artery size is due to reduction in aortic root pressure and not to changes in the left ventricle.

Left ventricular wall thickness decreased significantly after repair, suggesting that left ventricular hypertrophy before repair was due to high left ventricular afterload. Further evidence of relief of left ventricular afterload was provided by serial electrocardiography: of the 5 patients whose LVWT was reduced postoperatively, 4 also had regression of left ventricular hypertrophy.

Left ventricular hypertrophy is accompanied by increased coronary blood flow, so flow per unit of mass in the hypertrophied left ventricle at rest is the same as that in normal myocardium [22, 23]. In dogs, the size of epicardial coronary arteries increases concomitantly with increasing flow, but not proportionately; that is, 77% increase in left ventricular mass after banding of the ascending aorta is accompanied by only a 25% increase in cross-sectional area of epicardial arteries [24]. Whether coronary artery size decreases after resolution of left ventricular hypertrophy has not been previously documented. If our finding of lack of change in diastolic diameter of the coronary arteries concomitant with postoperative reduction in left ventricular hypertrophy is confirmed by a larger set of observations, this would suggest that there is little relation between coronary size and ventricular hypertrophy in the human as well as in the canine myocardium.

Conclusion

This study has several weaknesses. It was retrospective, so the data were not complete for all patients, and the variables examined were uncontrolled. There may have been a selection bias in choosing type of operation. All operations, however, were performed by only 2 surgeons using similar techniques throughout the 10-year period surveyed. The study groups were small, thereby weakening the power of the statistical analysis; thus, the negative findings, like the lack of difference in hemodynamic results after single sinus versus bisinus patch repair, rest on insecure ground. Nevertheless, the statistically significant positive findings in these small groups were meaningful.

We are able to conclude that repair of supravalvar aortic stenosis (localized and diffuse) by both single-sinus and bisinus patch repair is safe and hemodynamic results are good. The LCA diameter is larger than normal before and after operation, and LCA enlargement is greater than RCA enlargement preoperatively. Left ventricular hypertrophy and wall thickness regress after reparative operation.

We are grateful for the expert assistance of Martha Stroud, MS in Biometry, in performing the statistical analyses and of Kathryn Terry in preparing the manuscript.

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Appendix 1.

Normal values for the diameters of the aortic annulus and the descending thoracic aorta (DAo) during systole were derived according to body surface area from power function equations [5]. Body surface area data were converted to age-specific data using a standard nomogram for those two variables [7]. Normal values for right (RCA) and left coronary artery (LCA) diameters during systole were obtained from age-related data [6]. The coronary artery diameters were measured 0.5 cm from the vessels' ostia to be congruent with published normative data.

The age-related diameters (in millimeters) of the RCA, LCA, and DAo during systole were calculated to be:

Age	RCA	LCA	DAo
0–3 months	2.0	. . .	6.5
3–8 months	2.1	2.2	8.0
1–2.9 years	2.4	2.1	10.0
3–5.9 years	2.2	3.0	12.0
6–10 years	2.8	3.4	14.5
11–16 years	3.0	3.6	16.5

The derived normal values for RCA index and LCA index during systole were:

Age	RCA/DAo	LCA/DAo
0–3 months	0.31	. . .
3–8 months	0.26	0.28
1–2.9 years	0.24	0.21
3–5.9 years	0.18	0.25
6–10.9 years	0.20	0.23
11–16 years	0.18	0.22

DISCUSSION

DR ROBERT A. GUSTAFSON (Morgantown, WV): I thank Braunstein and associates for allowing me to review the manuscript and congratulate them on a very good presentation of an excellent series of patients with a difficult surgical problem.

Your observations about the changes in coronary artery size and LVWT after operation are very intriguing. You are also to be highly complimented on the management of the 2 patients with diffuse supravalvar aortic stenosis. The late incidence of aortic insufficiency is a little concerning, but I agree with the comments in the manuscript that this may be due to the sensitivity of echocardiography. I noted that a majority of the patients were symptomatic and the mean age at operation was 15 years. Do you think that age at operation has anything to do with the changes in coronary artery size, LVWT, or aortic valve competence? If we operated much earlier on asymptomatic patients, would these changes be less?

DR JAMES R. STEWART (Nashville, TN): My associates and I have recently reviewed our experience with this lesion at Vanderbilt, and we congratulate Braunstein and associates on their excellent results. Our results are very confirmatory of one of the conclusions they made about the value of the single-patch repair.

In the last 15 years at Vanderbilt, we have operated on 21 patients with supravalvar aortic stenosis and have had no operative deaths. All of the patients had single-patch repair, and we have seen two familial clusters of patients with Williams syndrome.

Three patients have required reoperation for valvar and subvalvar aortic stenosis and a complex arch anomaly, much like the patient you alluded to earlier, but otherwise all patients except 1 had effective relief of the gradient. Five patients in our group have associated peripheral pulmonary stenosis, and we believe that single-patch repair is very effective for this lesion, as you mentioned.

DR BRAUNSTEIN: There was no correlation between the ages of the 5 patients who underwent cardiac catheterization and the changes in their coronary artery morphometrics. There was also no correlation of age with pressure gradient or left ventricular hypertrophy. The lack of correlation in these variables, however, is not very meaningful because of the small number of patients with adequate data to make the comparisons.

Strategy for Lymphadenectomy in Lung Cancer Three Centimeters or Less in Diameter

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We have surgically treated 221 patients with a primary lung cancer 3.0 cm or less in diameter. There were 8 patients with a tumor 1.0 cm or less in diameter (group A), 84 with a tumor 1.1 to 2.0 cm in diameter (group B), and 129 with a tumor 2.1 to 3.0 cm in diameter (group C). The incidence of N0, N1, and N2 disease was 100%, 0%, and 0%, respectively, for patients in group A; 83%, 5%, and 12% in group B; and 62%, 12%, and 25% in group C (N0 versus N2, $p < 0.01$). For the 63 patients with regional lymph node involvement, "skipping" metastasis was present in 28.6%. The 5-year survival rate was 80% for group A, 74% for group B, and 51% for group C (group B versus group C, $p < 0.01$). Of the 121 patients who underwent complete resection and were followed up for 5 years, 41% had recurrence, 8% with local recurrence and 33% with distant metastasis. Therefore, it is important to do as complete a resection as possible together with mediastinal lymphadenectomy. Efforts to detect systemic spread should be made at the time of preoperative evaluation, even when the lung tumor is small.

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The rate of early detection of primary carcinoma of the lung has improved with progress in diagnostic techniques such as computed tomography, bronchoscopy, mediastinoscopy, and sputum cytology. These techniques have been used for preoperative TNM staging (denoting the importance of tumor size, nodal status, and spread of disease) [1]. There is little or no controversy over the preference for surgical intervention in the definitive management of T1 N0 M0 disease, such as the earliest lung cancer [2, 3]. However, in the management of a smaller tumor, and T1 disease in particular, the results are far from satisfactory, despite extended complete resections [4]. Even if the primary tumor is very small, massive pleural effusion, extensive mediastinal lymph node metastasis, or distant metastasis can be present [5].

For editorial comment, see page 691.

We report here prognostic factors in the management of patients with a tumor 3 cm or less in diameter and the importance of hilar and mediastinal lymph node dissection for these smaller tumors.

Material and Methods

During the 15-year period April 1974 to March 1989, 221 patients with lung cancers with a maximum tumor diameter of less than or equal to 3.0 cm underwent surgical resection in the Department of Surgery II, Faculty of Medicine, Kyushu University. Age of the patients ranged from 23 to 86 years (mean age, 63 years). Male patients greatly outnumbered female patients (153/68). All patients had a physical examination, chest roentgenography, whole-body computed tomography, bronchoscopy, and bone scanning, and preoperatively all were considered to have operable disease.

Complete resection of the tumor by lobectomy or pneumonectomy combined with mediastinal lymphadenectomy was performed as the standard treatment whenever possible. Of the 221 patients, 193 underwent complete resection and 28, incomplete resection. The extent of surgical excision was as follows: lobectomy in 181, bilobectomy in 15, pneumonectomy in 13, and partial resection in 12. Resected specimens were histologically examined, and the extent of lymph node spread was recorded. All tumors reviewed were subdivided based on the current criteria of the World Health Organization [6]. One hundred thirty-seven patients (62.0%) had adenocarcinoma; 60 (27.1%), squamous cell carcinoma; 11 (5.0%), small cell carcinoma; 10 (4.5%), large cell carcinoma; and 3 (1.4%), adenosquamous cell carcinoma. The hilar and mediastinal lymph nodes were identified by the lymph node map for lung cancer [7]. The stage of the disease was classified according to the TNM classification of the Union Internationale Contre le Cancer [1]. There were 151 patients with stage I, 14 with stage II, 36 with stage IIIA, 11 with stage IIIB, and 9 with stage IV disease.

One hundred twenty-one patients who had a complete resection were followed up for 5 years or until death for up to 5 years. Chest roentgenography, whole-body computed tomography, and bone scanning were done. In addition, specific attention was directed to the first site of

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recurrence. Local recurrence was defined as any recurrent disease within the ipsilateral hemithorax, mediastinum, or supraclavicular lymph nodes. All other sites of recurrence were considered to be distant metastases.

Survival rate was calculated by the Kaplan-Meier life-table method [8]. Statistical comparisons were made using the χ^2 test and the generalized Wilcoxon test [9].

Results

The 221 patients with a primary tumor 3.0 cm or less in diameter were separated into three subsets: group A, 8 patients (3.6%) with a tumor with a maximum diameter of less than or equal to 1.0 cm; group B, 84 patients (38.0%) with a tumor 1.1 to 2.0 cm in diameter; and group C, 129 patients (58.4%) with a tumor 2.1 to 3.0 cm in diameter. Variables assessed included sex, histology, T (tumor) status, N (node) status, M (metastasis) status, stage, and curability of operation (Table 1).

Table 1. Summary of Patient Data[a]

		Tumor Size (cm)		
Variable	No. of Patients	≤1.0 (group A)	1.1–2.0 (group B)	2.1–3.0 (group C)
Sex				
Male	153 (69)	6 (75)	57 (68)	90 (70)
Female	68 (31)	2 (25)	27 (32)	39 (30)
Histology				
Adenocarcinoma	137 (62)	3 (38)	55 (66)	79 (61)
Squamous cell	60 (27)	5 (63)	19 (23)	36 (28)
Adenosquamous cell	3 (1)	0	2 (2)	1 (1)
Large cell	10 (5)	0	3 (4)	7 (5)
Small cell	11 (5)	0	5 (6)	6 (5)
T status				
T1	191 (86)	8 (100)	81 (96)	102 (79)
T2	10 (5)	0	1 (1)	9 (7)
T3	11 (5)	0	0	11 (9)
T4	9 (4)	0	2 (2)	7 (5)
N status				
N0	158 (72)	8 (100)	70 (83)	80 (62)
N1	19 (9)	0	4 (5)	15 (12) [b]
N2	42 (19)	0	10 (12)	32 (25)
N3	2 (1)	0	0	2 (2)
M status				
M0	212 (96)	8 (100)	81 (96)	123 (95)
M1	9 (4)	0	3 (4)	6 (5)
Stage				
I	151 (68)	8 (100)	69 (82)	74 (57)
II	14 (6)	0	3 (4)	11 (9) [b]
IIIA	36 (16)	0	7 (8)	29 (23)
IIIB	11 (5)	0	2 (2)	9 (7)
IV	9 (4)	0	3 (4)	6 (5)
Curability of operation				
Complete resection	193 (87)	8 (100)	77 (92)	108 (84)
Incomplete resection	28 (13)	0	7 (8)	21 (16)
Total	221	8	84	129

[a] Numbers in parentheses are percentages. [b] Significance: $p < 0.01$.

Based on postsurgical evaluations, all patients in group A had T1 N0 M0 disease, the earliest form of lung cancer. Of the 84 patients in group B, 68 (81%) had T1 N0 M0 disease. One had involvement of the main bronchus (T2 disease) and 2, pleural metastases (T4 disease); 4 had only hilar lymph node involvement (N1 disease) and 10, mediastinal lymph node involvement (N2 disease). Three had distant metastasis (M1 disease). Of the 129 patients in group C, there were 70 patients (54%) with T1 N0 M0 disease. Six patients had pleural metastases, and 1 patient had direct invasion to the pulmonary artery (T4 disease); 15 had only hilar lymph node involvement and 34, mediastinal lymph node involvement (N2 and N3); 6 had distant metastasis. The rate of regional lymph node metastasis in patients with a primary tumor 3.0 cm or less in diameter increased with increase in tumor size. There was a significant difference in tumor size between patients with N0 disease and those with N2 disease ($p < 0.01$).

The frequency of lymph node metastasis was analyzed in the 63 patients (28.5%) with nodal involvement. The extent of involvement was as follows: 19 had hilar lymph node involvement and 44, mediastinal lymph node involvement. The level of the diseased lymph nodes in each of the 63 patients is shown in Figure 1. Of interest is the fact that 18 patients (28.6%) had "skipping" metastases, namely, involved lymph nodes at the mediastinal level but not at the hilar level.

The frequency of mediastinal lymph node metastasis was also examined in terms of the location of the tumors. Of the 73 lesions in the right upper lobe, 18 (24.7%) involved mediastinal lymph nodes. The incidence of diseased lymph nodes versus all tumors in the same lobe was as follows: 3%, highest mediastinal; 10%, paratracheal; 22%, pretracheal; 21%, tracheobronchial; and 6%, subcarinal. A tumor in the right upper lobe metastasized more frequently to the superior mediastinal lymph nodes.

Of the 56 lesions in the left upper lobe, 9 (16.1%) involved mediastinal lymph nodes. The incidence of diseased lymph nodes was as follows: 4%, pretracheal; 7%, tracheobronchial; 7%, subaortic, 5%; paraaortic; and 5%, subcarinal. A tumor in the left upper lobe tended to metastasize to the aortic lymph nodes.

Of the 67 lesions in the right middle and lower lobes, 14 (20.9%) involved mediastinal lymph nodes. The incidence of diseased lymph nodes was as follows: 3%, highest mediastinal; 5%, paratracheal; 6%, pretracheal; 8%, tracheobronchial; 13%, subcarinal; 3%, paraesophageal; and 2%, pulmonary ligament.

Of the 25 lesions in the left lower lobe, 3 (12.0%) involved the mediastinal lymph nodes. The incidence of diseased lymph nodes was as follows: 4%, pretracheal; 4%, tracheobronchial; and 12%, subcarinal. Both the right middle and lower lobes and the left lower lobe showed a tendency to metastasize more frequently to the subcarinal lymph nodes.

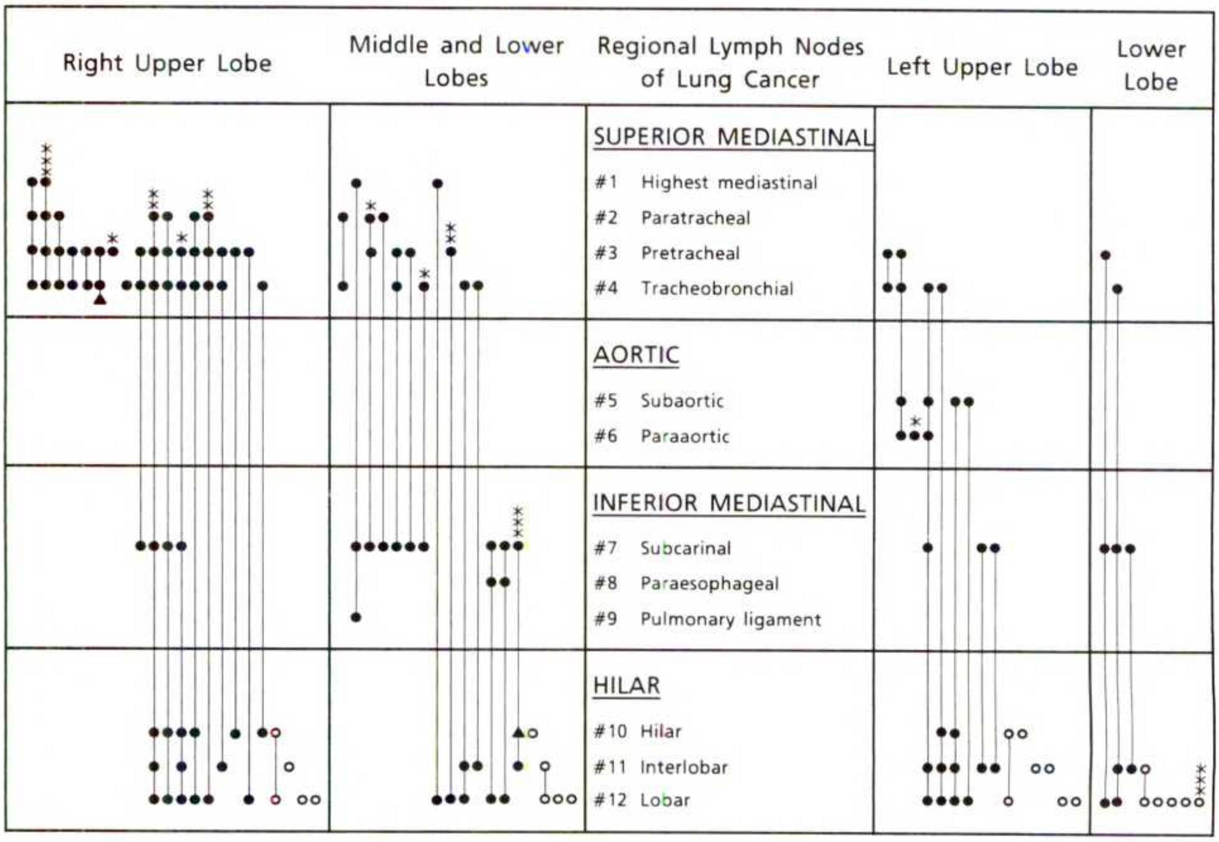

*Fig 1. Location of diseased nodes in 63 patients with regional lymph node involvement: mediastinal lymph node involvement (N2 disease) (●——●); contralateral hilar or mediastinal lymph node involvement (N3 disease) (▲); and hilar lymph node involvement (N1 disease) (○——○). (No asterisk = adenocarcinoma; * = squamous cell carcinoma, ** = large cell carcinoma; *** = small cell carcinoma.)*

The survival rate at 5 years was 61% for all 221 patients with a primary tumor 3.0 cm or less in diameter, 65% for the 193 patients having complete resection, and 39% for the 28 patients having incomplete resection ($p < 0.05$). Survival curves were drawn for the three groups (Fig 2). The 5-year survival rate was 80% for group A, 74% for group B, and 51% for group C. The smaller the diameter of the lesion, the longer the survival. There was a significant difference between the findings in groups B and C ($p < 0.01$). Survival rates were determined according to the various prognostic factors (Table 2). There were significant differences in the findings between N0 versus N1 disease and N0 versus N2 disease ($p < 0.01$). The survival of patients with a tumor in the left lower lobe was shorter than that of patients with a right upper lobe lesion ($p < 0.01$).

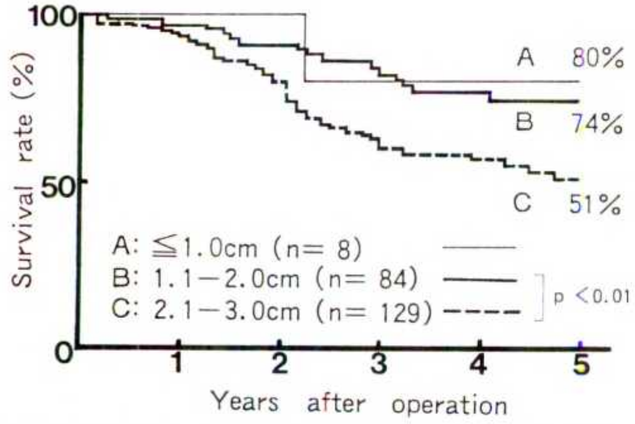

Fig 2. Survival curves for patients with a primary tumor 3.0 cm or less in diameter by subset of tumor size.

Fifty (41%) of the 121 patients who underwent complete resection and were followed up for 5 years or until death for up to 5 years had recurrences: 10 (8%), local, and 40 (33%), distant metastases. The sites of the first recurrence are shown in Table 3. Of the ten local recurrences, seven were adenocarcinomas and one each was squamous cell carcinoma, large cell carcinoma, and adenosquamous cell carcinoma. Of the 40 distant metastases, 20 were adenocarcinomas, 13 were squamous cell carcinomas, 4 were large cell carcinomas, and 3 were small cell carcinomas. Adenocarcinoma tended to recur locally and to metastasize widely. Of the patients with local recurrence, 5 had N0 disease, 2 had N1 disease, and 3 had N2 disease. Of the patients with distant metastasis, 23 had N0 disease, 6 had N1 disease, and 11 had N2 disease. Neither local

Table 2. Survival Rates According to Various Prognostic Factors

Factor	No. of Patients	Survival Rate (%)				
		1 Year	2 Years	3 Years	4 Years	5 Years
Sex						
Male	153	94	81	69	63	60
Female	68	100	87	73	71	65
Histology						
Adenocarcinoma	137	98	84	72	65	62
Squamous cell	60	94	79	69	66	62
Adenosquamous cell	3	100	100	50	50	0
Large cell	10	79	79	79	79	79
Small cell	11	100	88	58	58	58
T status						
T1	191	97	87	74	68	66 [a]
T2	10	100	73 [a]	57	57	19
T3	11	83	65	52	52	52
T4	9	89	42	42	42	. . .
N status						
N0	158	99 [a]	94 [a]	82 [a]	77 [a]	75 [a]
N1	19	78	56 [a]	44 [a]	32 [a]	25 [a]
N2	42	95	60	42	42	34
N3	2	33	. . .	. . .	. . .	. . .
M status						
M0	212	96	84	71	67 [b]	63
M1	9	100	63	47	28	28
Stage						
I	151	100 [b]	94 [a]	82 [a]	79 [a]	76 [a]
II	14	78	54 [a] [a]	46 [b]	28 [a]	17 [a]
IIIA	36	91	68	47	47 [a]	40 [b]
IIIB	11	79	43	43	43	. . .
IV	9	100	62	47	28	28
Location of lesion						
Right upper	73	94	78	72	69	69
Right middle and lower	67	95 [a]	89	78	73 [a]	66 [a]
Left upper	56	100	87	67	63 [a]	54
Left lower	25	96	71	53	41	41
Curability of operation						
Complete resection	193	98	87 [a]	74 [b]	69 [a]	65 [b]
Incomplete resection	28	85	56	46	39	39
Total	221	96	83	70	65	61

[a] Significance: $p < 0.01$. [b] Significance: $p < 0.05$.

recurrence nor distant metastasis was affected by nodal status at the time of operation.

Comment

The most important finding in the present study was that the actuarial 5-year survival rate for patients with a small lung cancer decreased with increase in tumor size. This can be explained by an increase in the incidence of lymph node metastasis at the time of operation or skipping metastasis. The mode of lymphatic metastasis was affected by tumor size and location of the tumor but not by other factors, including sex and tumor histology.

Soorae and Abbey-Smith [10] reported that the larger the lung tumor, the worse the prognosis. Five-year survival was 60.0% for patients with a tumor less than 3 cm in diameter, 35.6% for those with a tumor 3.0 to 4.9 cm, 18.0% for those with a tumor 5.0 to 6.9 cm, and 4.5% for those with a tumor 7 cm in diameter or greater. Jackman and co-workers [11] stated that a small tumor was a highly

Table 3. Sites of First Recurrence in 50 of the 121 Patients With Complete Resection and 5-Year Follow-up

		Tumor size (cm)		
Recurrence	No. of Patients	≤1.0 (group A)	1.1–2.0 (group B)	2.1–3.0 (group C)
Local recurrence				
Mediastinal lymph node	6	0	3	3
Supraclavicular lymph node	3	0	1	2
Pleura	1	0	1	0
Total	10	0	5	5
Distant metastasis				
Lung	20	0	6	14
Bone	9	0	3	6
Brain	9	1	3	5
Liver	1	0	0	1
Adrenal gland	1	0	0	1
Total	40	1	12	27

favorable prognostic indicator, 5-year survival being 68.2% for patients whose lesion measured 2.0 cm or less. In 1988, we [2] reported that the 5-year survival rate of patients with lung cancer less than 2.0 cm in diameter was 76%. Shields and co-workers [12] considered the presence or absence of lymph node metastasis to be a prognostic factor after resection of lung cancer; 5-year survival was 20.1% for patients with lobar node involvement, 17.4% for those with hilar node involvement, and 8.9% for those with mediastinal node involvement. In the present study we also found that small lung cancers tend to progress to nodal involvement as the tumor enlarges, and the prognosis is poor.

Lymph node metastasis has been found to occur frequently in patients with lung cancer, the incidence being 50% to 60% in those undergoing resection [13, 14]. We noted lymph node metastasis in 29% of patients with a small tumor: 0% of patients with a tumor less than 1.0 cm in diameter, 17% with a tumor 1.1 to 2.0 cm in diameter, and 38% of those with a tumor 2.1 to 3.0 cm in diameter. Among the patients in whom metastasis to lymph nodes was noted at the time of operation, 28.6% had skipping metastasis.

Autopsy data on 202 patients with lung cancer who were treated by complete resection and died within 30 days of operation showed that 35% had persistent disease [15]. Here, 50% had disease limited to the bronchial stump or to the hilar or mediastinal lymph nodes, and the remainder had a distant metastasis. In other studies involving resection in patients with stage I or II disease, approximately 60% had a distant metastasis and 40%, a local recurrence [16, 17]. The lung and brain were the most frequent sites of the first recurrence, and the rates of relapse were approximately 40% and 20%, respectively [4, 17]. The rates of recurrence were higher for patients with adenocarcinoma and large cell carcinoma compared with those with squamous cell carcinoma [18]. We found that the rates of local recurrence and distant metastasis were higher with adenocarcinoma.

It is apparent that recurrence, regional and distant, is more common than previously recognized in patients with a small tumor, even in the absence of lymphatic metastasis [2, 4, 5, 16, 17]. These recurrences suggest that malignant cells are present in regional lymph nodes and distant organs at the time of preoperative evaluation and staging. Based on the morphological evidence of N2 disease, the incidence of patients with microscopic involvement was 29%, and the survival rate was higher than that of patients with gross involvement of mediastinal lymph nodes [19]. Moreover, skipping metastasis in regional lymph nodes was frequent in patients with lung cancer. For patients with a small malignant tumor in the lung, dissection of mediastinal lymph nodes should be done, even for those in whom metastasis to regional lymph nodes is not apparent at the time of resection [20, 21].

In conclusion, we emphasize that regional lymph node metastasis is not rare, even in patients with a tumor 1.0 to 3.0 cm in diameter, and we recommend complete resection with mediastinal lymphadenectomy for local control of the disease. Extra efforts to define systemic spread are justified even if the preoperative evaluation reveals no distant metastasis. Adjuvant chemotherapy for patients with a small malignant tumor of the lung requires further study.

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Choreoathetosis After Deep Hypothermia Without Circulatory Arrest

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In 8 of 758 patients undergoing an intracardiac operation under cardiopulmonary bypass and hypothermia, choreoathetosis developed 3 to 7 days postoperatively. Before the onset of choreoathetosis, varying degrees of neurological dysfunction were noted. Electroencephalography and neuroimaging failed to detect any responsible functional or structural changes. Six patients are alive 1 to 3 years postoperatively, and their condition is improving. Two patients died of aspiration or sepsis. All patients were grouped based on factors identified as being possibly causative: depth of hypothermia, cooling time, flow rate, and repeated hypothermia. The incidence of choreoathetosis was significantly different in group A (rectal temperature $>25°C$) compared with group B (rectal temperature $\leq 25°C$) (0/295 versus 8/463; $p = 0.02$). Based on cooling time, the incidence of choreoathetosis was significantly different in group B_1 (cooling time < 1 hour) compared with group B_2 (cooling time ≥ 1 hour) (1/220 versus 7/243; $p = 0.05$). Based on flow rate during cooling, group B_2 was further divided into the low-flow group ($<1{,}500\ mL \cdot min^{-1} \cdot m^{-2}$) and the high-flow group ($\geq 1{,}500\ mL \cdot min^{-1} \cdot m^{-2}$). Although not significant, the incidence of choreoathetosis was higher in the high-flow group (6/153 versus 1/90; $p = 0.22$). In group B patients having reoperation, the incidence of choreoathetosis was higher than in patients operated on for the first time (5/54 versus 3/409; $p \leq 0.0001$). Our data suggest that deep hypothermia of 25°C or lower along with a cooling time of 1 hour or longer, maintenance of a high flow rate, and repeated exposure to hypothermia may predispose to the development of choreoathetosis postoperatively.

(Ann Thorac Surg 1990;50:714–9)

The development of choreoathetosis after an open heart operation in children remains an enigma [1, 2]. Electroencephalography and neuroimaging often fail to detect any functional or structural abnormality in contrast to the findings for many other early postoperative neurological problems such as embolism, cerebral infarction, and intracranial bleeding [3, 4].

Several possible mechanisms of brain injury causing choreoathetosis have been proposed, including anoxia during circulatory arrest, uneven cooling, no-reflow phenomenon, hyperglycemia, and excessive dopaminergic activity [2, 5–13]. The lack of a consistent factor associated with choreoathetosis makes prevention quite difficult and results in continued occurrence of this dreadful postoperative neurological event.

For editorial comment, see page 693.

Over a 3-year period, we encountered several patients in whom choreoathetosis developed postoperatively. Therefore, we undertook a retrospective study to find a common responsible factor by analyzing these patients and all other patients undergoing an intracardiac operation during the same period.

Material and Methods

From June 1986 to April 1989, choreoathetosis developed postoperatively in 8 of 758 patients who survived an intracardiac operation under cardiopulmonary bypass and hypothermia. To determine the possible cause or causes of the choreoathetosis in these patients, their clinical profiles, anesthesia records, operative notes, and cardiopulmonary bypass records were reviewed. The findings in these 8 patients were compared with those of the other 750 patients using unpaired Student's *t* test and χ^2 analysis for tests of significance.

A summary of the clinical data on the patients in whom choreoathetosis developed is shown in Table 1. Although none of the patients had seizures or gross neurological deficits preoperatively, some had additional complicating abnormalities. Patient 1 had a congenital right facial nerve palsy and an abnormal right ear with absent auditory canal. A computed tomographic scan of the brain showed prominent cerebrospinal spaces, a finding suggesting mild cerebral atrophy. Patient 6 had had *Haemophilus*-caused sepsis with mediastinal involvement and meningitis a year before the last operation. A brain computed tomographic scan showed two small areas of infarction.

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Table 1. Summary of Clinical Data on 8 Patients With Choreoathetosis

Patient No.	Age (y)	Diagnosis	Previous Operation	Most Recent Operation
1	2	DORV, TAPVD, pulmonary atresia, asplenia, PVO	Cooley shunt (newborn); repair of TAPVD (3 mo)	Relief of PVO
2	5	TGA, VSD, RV dysfunction	Mustard, VSD closure (14 mo); PAB (2 y)	Takedown of Mustard, arterial switch
3	5	Tetralogy, absent pulmonary valve	Subclavian–pulmonary shunt and MPA ligation (newborn)	Repair
4	3	CAVC, decreased LV function, subaortic stenosis	PAB (2 mo)	MPA–aorta anastomosis; Glenn shunt
5	2	Truncus arteriosis, truncal valve insufficiency, conduit stenosis	Repair (6 mo)	Truncal valvoplasty; conduit replacement
6	4	TGA, VSD, conduit stenosis	Subclavian–pulmonary shunt (6 mo); Rastelli (2 yr)	Conduit replacement
7	2.5	VSD	None	Closure
8	1.2	VSD, PVO	PAB (1 mo); VSD closure (10 mo)	Relief of PVO

CAVC = common atrioventricular canal; DORV = double-outlet right ventricle; LV = left ventricular; MPA = main pulmonary artery; PAB = pulmonary artery banding; PVO = pulmonary venous obstruction; RV = right ventricular; TAPVD = total anomalous pulmonary venous drainage; TGA = transposition of great arteries; VSD = ventricular septal defect.

Patient 8 had recurrent pulmonary infections or edema or both necessitating intubation and long-term ventilatory support since ventricular septal defect closure and pulmonary artery debanding at 10 months of age. Surgical intervention this time consisted of repair of bilateral pulmonary venous obstruction at age 15 months. Five patients had undergone previous intracardiac procedures under cardiopulmonary bypass and deep hypothermia.

Anesthesia

Anesthesia induction was carried out by administration through a mask of an inhalation anesthetic (Forane [isoflurane] or halothane) and oxygen. Ketamine hydrochloride was administered intramuscularly to 4 patients as an induction agent. On insertion of an intravenous line, pancuronium bromide was given, followed by endotracheal intubation. Anesthesia was maintained with the inhalation anesthetic, intermittent intravenous administration of Sublimaze (fentanyl citrate), and a paralyzing dose of pancuronium. No unusual event was noted during induction in any of the 8 patients. Antibiotics and methylprednisolone (20 mg/kg) were also given intravenously.

Surgical Technique

Indications for operation are listed in Table 1. Operations were carried out through a median sternotomy incision, with 5 patients undergoing uneventful repeat sternotomy. Cannulation and cardiopulmonary bypass were accomplished through the ascending aorta and right atrium. A Terumo hollow-fiber oxygenator, a Sarns dual heater-cooler, and a roller pump were used. A sump was used in 4 patients (through the right superior pulmonary veins in 2 and through the left ventricular apex in 2); for the other 4 patients, there was no mention of a sump in the operative note. Blood cardioplegia was given to 5 patients, whereas in the other 3 patients, the operation was performed with the heart in ventricular fibrillation. The blood cardioplegic solution was prepared by filling up the cardioplegia pump reservoir with the cardiopulmonary bypass perfusate and adding 20 mEq of KCl, 3 g of mannitol, 200 μg of nitroglycerin, and 12.5 g of albumin per liter.

Cardiopulmonary Bypass and Hypothermia

Core cooling was accomplished through cardiopulmonary bypass and the heat exchanger using the rectal temperature to monitor the depth of cooling. Esophageal temperature was used as a measure of blood perfusate temperature. A gradient of no more than 10°C between the water and esophageal temperatures was maintained during cooling and rewarming.

All patients were cooled to a rectal temperature of 25°C or lower (mean temperature, 21° ± 3°C; range, 16° to 25°C) with an esophageal temperature of 22°C or lower (mean temperature, 19° ± 2°C; range, 16° to 22°C). While on cardiopulmonary bypass, arterial and venous blood gases taken at 15-minute and 30-minute intervals, respectively, were uneventful. The length of time the blood perfusate temperature was kept at 25°C or lower (cooling time) ranged from 30 to 179 minutes (mean time, 91 ± 46

Table 2. Cardiopulmonary Bypass and Hypothermia

Patient No.	Temperature (°C) (Rectal/ Esophageal)	Bypass Time (min)	Cooling Time (min)	Flow ($mL \cdot min^{-1} \cdot m^{-2}$)
1	16/16	155	110	1,500 × 35 min Low flow × 45 min
2	20/20	270	179	1,500
3	25/16	189	120	1,990
4	21/21	90	60	2,200
5	20/19	180	78	2,000
6	20/19	120	60	2,500
7	25/22	75	30	1,900
8	21/20	240	89	1,500

minutes). Except for 1 patient who had a short cooling time of 30 minutes, the rest had 60 minutes or longer.

The rate of blood flow while the blood perfusate was 25°C or lower was also analyzed. Normal blood flow was calculated at 2,000 to 2,500 $mL \cdot min^{-1} \cdot m^{-2}$. In 7 patients, blood flows ranged from 1,500 to 2,500 $mL \cdot min^{-1} \cdot m^{-2}$ (mean flow, 1,941 ± 360 $mL \cdot min^{-1} \cdot m^{-2}$) during the cooling period. In the remaining patient, rectal and esophageal temperatures were 16°C and total cooling time was 110 minutes; however, the blood flow was maintained at 1,500 $mL \cdot min^{-1} \cdot m^{-2}$ or higher for only 35 minutes with the flow being kept as low as 300 $mL \cdot min^{-1} \cdot m^{-2}$ the rest of the time. Total bypass time in these 8 patients ranged from 75 to 270 minutes (mean time, 165 ± 69 minutes). Temperatures, bypass times, cooling times, and blood flows are detailed in Table 2.

Results

Clinical Findings and Outcome

The extubation day, early mental state, onset of choreoathetosis, neurological test results, and outcome are outlined in Table 3. Seven patients were extubated within 24 hours after the operation, although 1 required reintubation because of agitation and pulmonary edema. This patient was successfully extubated on the fifth postoperative day. The eighth patient, who had had tracheostomy and long-term respiratory support preoperatively, remained on the respirator until the patient died of pneumonia and sepsis 2 months later.

The full syndrome of choreoathetosis developed in all patients 3 to 7 days after the operation and consisted of seemingly purposeless continued movements of head, torso, and all extremities while awake but not while asleep. All patients became unable to swallow, support the head, or walk once the full syndrome appeared. Early

Table 3. Clinical Course

Patient No.	Time of Extubation	Early Mental State[a]	Onset of Choreoathetosis (POD)	Tests[a]	Outcome
1	POD 1	Agitated, disoriented	3	EEG (2), MRI (7): normal	Aspirated at home; died (1 mo)
2	POD 1, 5[b]	Agitated, behavior inappropriate	7	EEG (7, 10): slow waves; CT (7): normal	Mild learning disability (2 y)
3	POD 1	Alert, talking, walking	7	EEG (11), CT (11): normal	Mild choreiform movements; otherwise normal (2 y)
4	POD 1	Alert, appropriate behavior (2); agitated, mumbling	5	EEG (9), CT (3), LP (4), MRI (22): normal	High-stepping gait, talks well (3 y)
5	OD	Awake, calm (1); talked to mother (2); "spacey" (3)	4	EEG (5), CT (6), LP (7): normal	Improving (14 mo)
6	OD	Awake, oriented, irritable (1); "spacey" (2)	4	EEG (6): normal; CT (3): old infarcts	Improving (12 mo)
7	OD	Sleepy (OD); alert, up and about (1); irritable (3)	7	EEG (8)[c]; CT (8), MRI (60): normal	Improving (13 mo)
8	Not accomplished	Seizures (1–3)	6	EEG (1): seizures; ultrasound (1): cerebral atrophy	Died of pneumonia and sepsis (2 mo)

[a] Numbers in parentheses are postoperative days. [b] This patient was extubated and reintubated on POD 1 for pulmonary edema and extubated on POD 5. [c] The EEG was normal except for rare spike in left frontal and parietal regions.

CT = computed tomography; EEG = electroencephalogram; LP = lumbar puncture; MRI = magnetic resonance imaging; OD = operative day; POD = postoperative day.

signs of neurological dysfunction were observed a few days before the onset of choreoathetosis. Five patients were agitated with varying degrees of disorientation. Two patients were noted to follow with the eyes without moving the head. Drooling was noted in 3 patients. One patient did not show irritability but was described as being "out of it" or "spacey." One patient (patient 8), who was chronically ill, had seizures before the onset of choreoathetosis. Only 1 patient (patient 3) appeared to have no early neurological deficit; she was eating, talking, and walking until the seventh postoperative day when she stopped communicating and the choreoathetoid movements started.

Electroencephalography was performed in all patients. The results were completely normal in 5 patients, showed a rare spike in the left frontal and parietal regions in 1 patient, and showed a slow wave in another patient. Patient 8 was the only patient who showed definite seizure activity. A computed tomographic scan of the brain in 6 patients was normal except for old infarcts in 1 patient (patient 6), who had had previous *Haemophilus* meningitis. Magnetic resonance imaging of the brain, done in 3 patients, was normal. A brain ultrasound in patient 8 showed cerebral atrophy. Lumbar puncture in 2 patients was equally unrevealing.

Six patients are alive 1 to 3 years after the development of choreoathetosis. One patient who has been followed up for 2 years is in school and, except for a minor learning disability, is normal. Two patients followed up for 2 to 3 years have minimal choreiform movements and are able to communicate and walk fairly well, although a high-stepping gait is present in 1. The 3 most recent patients, who have been followed up for 12 to 14 months, are undergoing rehabilitation and are showing varying degrees of improvement in speech, ability to eat, ambulation, and choreoathetoid movements.

Two patients died. One patient, who was sent home and required nasogastric feeding, died of aspiration 1 month postoperatively. The other patient, who remained on the respirator postoperatively, died of pneumonia and sepsis 2 months after operation.

Depth of Cooling, Cooling Time, and Rate of Blood Flow

All patients who survived for 30 days after an intracardiac procedure under cardiopulmonary bypass and hypothermia were categorized according to degree of cooling ($>$ 25°C and $\leq$ 25°C), duration of cooling time ($<$ 1 hour and $\geq$ 1 hour), and flow rate during cooling ($\geq$ 75% and $<$ 75% of minimum calculated blood flow).

Based on depth of hypothermia, all patients were divided into two groups: group A (rectal temperature $>$ 25°C; n = 295) and group B (rectal temperature $\leq$ 25°C; n = 463). Group A had a mean age of 5 ± 5 years and group B, 2.6 ± 4 years ($p \leq 0.0001$). The temperature in group A ranged from 25.2° to 35°C with a mean of 29° ± 2°C versus a range of 16° to 25°C with a mean of 23° ± 3°C in group B ($p \leq 0.0001$). In a small number of patients in whom the temperature recorded was in Fahrenheit, conversion to Celsius resulted in fractions. Mean bypass time in group A was 80 ± 44 minutes versus 144 ± 78 minutes in group B ($p \leq 0.0001$).

The incidence of choreoathetosis was 8 of 463 group B patients versus none of 295 group A patients ($p = 0.02$). In addition, when bypass perfusate or esophageal temperature was considered, the 8 patients in whom choreoathetosis developed belonged to the group of 273 patients who had a temperature of 22°C or less, versus none of 190 patients who had a temperature of greater than 22°C ($p = 0.02$).

Based on cooling time, group B patients were subdivided further into group B_1 (cooling time $<$ 1 hour; n = 220) and group B_2 (cooling time $\geq$ 1 hour; n = 243). The mean temperature in group B_1 was 24° ± 3°C versus 21° ± 3°C in group B_2 ($p = 0.0001$). The mean cooling time in group B_1 was 37 ± 13 minutes versus 93 ± 30 minutes in group B_2 ($p < 0.0001$). The incidence of choreoathetosis of 1 of 220 group B_1 patients versus 7 of 243 group B_2 patients proved to be significant ($p = 0.05$).

Because of the very suggestive correlation between depth of cooling of 25°C or lower and length of cooling time of 1 hour or longer, we thought that maintenance of a high rate of blood flow during cooling could also be contributory. Therefore, the incidence of choreoathetosis was further looked at in group B_2 patients who had a blood flow rate of 1,500 $mL \cdot min^{-1} \cdot m^{-2}$ or greater during the cooling period. Choreoathetosis developed in 6 of 153 patients in this group versus 1 of 90 patients who had a flow rate less than 1,500 $mL \cdot min^{-1} \cdot m^{-2}$. Although the incidence was higher in the high-flow group, the difference was not significant ($p = 0.22$).

The youngest patient in whom choreoathetosis developed was 1.2 years old. This prompted analysis of our group of infants 3 months old or younger (n = 70), all belonging to group B (rectal temperature $\leq$ 25°C), who underwent deeper levels of hypothermia (mean temperature, 19° ± 2°C; range, 15° to 25°C). There were 43 infants (61%) with transposition of the great arteries, 13 (18.5%) with total anomalous pulmonary venous drainage, 7 (10%) with hypoplastic left heart syndrome, 6 (8.5%) with ventricular septal defect, and 1 infant with critical aortic stenosis. In these infants, after the desired level of rectal temperature was reached, either circulatory arrest or 50% reduction of flow was done. The onset of bypass to arrest or reduction of flow ranged from 8 to 70 minutes (mean time, 22 ± 12 minutes), indicating a shorter cooling time; in only 9 infants did the time exceed 30 minutes, during which the flow rate was kept at 75% of calculated minimum flow. Total circulatory arrest was performed in 14 infants with the arrest period ranging from 5 to 65 minutes (mean time, 38 minutes). Excluding the infants with hypoplastic left heart syndrome, the arrest period ranged from 5 to 23 minutes (mean time, 13 minutes).

Previous Cardiopulmonary Bypass and Hypothermia

Because 5 of the 8 patients in whom choreoathetosis developed had undergone a previous intracardiac operation under hypothermia, the possible contributory effect of a previous exposure was examined. The 463 patients in group B (rectal temperature $\leq$ 25°C) were separated into those who had had previous intracardiac procedures (n =

54) and those undergoing an intracardiac operation for the first time (n = 409). The mean temperature was not significantly different between the two groups (23° ± 2°C versus 23° ± 3°C). All 5 patients having reoperation had been previously cooled to a rectal temperature of 17° to 23°C. The incidence of choreoathetosis in the group having reoperation proved to be significantly higher (5/54 versus 3/409; $p = 0.0001$).

Comment

Choreoathetosis remains a major postoperative problem in children undergoing intracardiac operation [1, 2]. The incidence has been estimated to range from 1% to 12% [14]. A recent survey [1] of six large pediatric cardiac centers showed continued postoperative occurrence of choreoathetosis.

Because most reports have associated the development of choreoathetosis with deep hypothermia and circulatory arrest, its pathogenesis has often been related to inadequate brain protection during the arrest period. Several investigators [15, 16] have shown varying degrees of metabolic and electrical activity in the brain that could lead to anoxic injury during circulatory arrest. A high cooling gradient or other factors favoring uneven brain cooling [6, 7] may exaggerate the brain anoxia during the arrest period. Other investigators [8, 9] have implicated uneven brain reperfusion as a result of vascular changes related to the "no-reflow phenomenon." Robinson and colleagues [2] suggested that if there was sufficient cellular damage to interrupt neurotransmitter synthesis or uptake along with increased dopaminergic activity, the intracellular stores of neurotransmitters such as acetylcholine would be depleted, leading to the delayed onset of choreoathetosis. Brunberg and co-workers [5] proposed that high tissue levels of glucose from hyperglycemia related to cardiopulmonary bypass might lead to increased glycolysis in the brain during arrest and result in injury from accumulated lactic acid metabolites.

There have been early clinical reports of brain damage, including choreoathetosis, from deep hypothermia without circulatory arrest. In 1962, Egerton and colleagues [17] reported that 15 of 16 adult patients who underwent profound hypothermia of 8° to 12°C esophageal temperature had mild to severe brain damage postoperatively. Findings included transient disorientation, retrograde amnesia, facial palsies and supranuclear palsies affecting eye movements and accommodation, generalized hypotonia and loss of reflexes, and even irreversible coma. A subsequent comparable series of patients operated on at an esophageal temperature of 24° to 30°C survived without any brain problems. Egerton and colleagues concluded that there is no indication to ever use hypothermia lower than 15°C.

Björk and Hultquist [18] reported brain damage, including choreoathetosis, in children undergoing hypothermia of 5.8° to 16°C esophageal temperature with and without circulatory arrest. Cortical ischemia, degeneration of cells of the basal ganglia and thalamus, and disappearance of Purkinje cells and myelin sheath were noted. Aggregation of thrombocytes and white blood cells were blamed for some of the changes.

Experimental evidence that hypothermia alone can cause brain damage has also been reported. Connolly and colleagues [19] demonstrated a decrease in the number of Purkinje cells in brains cooled to 17°C irrespective of perfusion techniques and clinical neurological damage. Molina and associates [20] also showed microscopic cellular damage in the brain of dogs subjected to a core temperature of 18°C with or without circulatory arrest. The brain, because of its high liquid content in contrast to other organs, might be more susceptible to certain levels of hypothermia.

Although most reports have associated the development of choreoathetosis to factors related to circulatory arrest rather than hypothermia, a closer look at these reports might suggest otherwise. Brunberg and colleagues [21] reported that choreoathetosis developed in 4 (18%) of 22 children after deep hypothermia (14° to 23°C nasopharyngeal temperature) and circulatory arrest. In the final minutes of cooling, the carbon dioxide was increased to 10% of the total gas flow, and the perfusate temperature (approximates esophageal temperature) was lowered to 4° to 10°C to provide maximal cerebral dilatation and cooling. Robinson and co-workers [2] saw 5 patients with choreoathetosis over an 18-month period. The period of circulatory arrest was rather short, ranging from 7 to 45 minutes, and the core temperature was low, ranging from 9° to 15°C. Castaneda and associates [22] reported that choreoathetosis developed in 9 (13%) of 70 infants after deep hypothermia (20°C rectally) and circulatory arrest. Hypothermia was accomplished through surface cooling to a rectal temperature of 25°C followed by core cooling at 100 $\text{mL} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ to 20°C. In a recent survey of six major pediatric cardiac centers using deep hypothermia (12° to 24°C), Ferry [1] found continued occurrence of choreoathetosis.

Thus, there is clinical and experimental evidence that hypothermia alone can produce brain damage, necessitating a need for reassessing the level of hypothermia employed in clinical practice. There is also a need for uniformity in monitoring body temperature during cooling. Three areas are commonly used: the esophagus, the nasopharynx, and the rectum. Because of the proximity of the esophagus to the aorta, the esophageal temperature often reflects the blood perfusate temperature and is the first to come down, followed by the nasopharyngeal and rectal temperatures. Depending on the speed of cooling, there could be a difference of a few degrees among the three temperatures during the early phase of cooling. Our experience indicates that at a rectal temperature of 25°C or lower, there is a 2% possibility (8/463) of encountering choreoathetosis in patients undergoing cardiopulmonary bypass without circulatory arrest. This projected incidence may increase to 3% (7/243) if cooling time is prolonged to 1 hour or longer, and to 4% (6/153) if pump flow during cooling remains high. The rectal temperature of 25°C is probably higher than the esophageal temperature of 16°C at which Björk and Hultquist [18] and Egerton and colleagues [17] found substantial brain damage. The depth of hypothermia at which Brunberg and colleagues [21], Robinson and co-workers [2], Castaneda and associates [22], and other investigators [23–25] have encountered choreoathetosis is probably comparable with ours; however, circulatory arrest was carried out in their patients.

Our experience suggests that a longer cooling time and the maintenance of a high rate of flow during cooling might be contributory to the development of choreoathetosis. This conclusion is supported by the findings of earlier investigators. Egerton and colleagues [17] found major brain damage in patients undergoing 134 minutes of cooling at 12°C but slight brain damage in patients undergoing 73 minutes of cooling ($p = 0.05$). They also suggested that high flow rates may be partly responsible for the brain damage. In their perfusion of canine heads at a rate greater than $10 \text{ mL} \cdot \text{min}^{-1} \cdot \text{kg}^{-1}$ at 17°C, Connolly and associates [19] universally produced brain damage.

The apparent higher incidence of choreoathetosis in patients who have had previous cardiopulmonary bypass and hypothermia raises the possibility of cumulative structural changes in the brain in spite of often normal electroencephalograms and neuroimaging. Experimental and clinical studies have shown microscopic changes in the brain secondary to hypothermia irrespective of symptoms [17–20].

In summary, our data suggest that deep hypothermia of 25°C or lower along with a cooling time of 1 hour or longer and the maintenance of a flow rate of 75% of calculated minimum flow or greater may predispose to the development of choreoathetosis postoperatively. Also, repeated exposure to hypothermia may have a cumulative effect neurologically and may further increase the incidence of choreoathetosis. We think that most congenital or acquired heart defects regardless of complexity can be safely operated on under cardiopulmonary bypass and hypothermia of 28°C. In smaller infants, the temperature can probably be safely lowered to 25°C provided attention is paid to shorter cooling time and maintenance of lower flow rates. Only a few infants, such as those with hypoplastic left heart syndrome, those in whom aortic arch reconstruction is needed, those with transposition of the great arteries with ventricular septal defect, and those with certain types of total anomalous pulmonary venous drainage, would probably require deeper levels of hypothermia and circulatory arrest.

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Prognostic Determinants in Extracorporeal Membrane Oxygenation for Respiratory Failure in Newborns

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Extracorporeal membrane oxygenation (ECMO) is becoming an accepted therapeutic modality for newborn respiratory failure, but there is little information available regarding the prognostic determinants with this technique. One hundred thirty-five newborns treated with ECMO over a 4-year period were critically analyzed with regard to the influence that birth weight, gestational age, age at initiation of ECMO, best blood gases before ECMO, number of hours on ECMO, renal failure, intracerebral hemorrhage, and long-distance air transport had on survival. Infants with meconium aspiration and those undergoing long-distance transfer showed significant differences in blood gases before ECMO, with survivors having more normal pH and carbon dioxide tension values. Intracerebral hemorrhage and renal failure that developed during ECMO were grave prognostic signs, with few survivors in either group. These data show that ability to ventilate patients before ECMO, giving normal carbon dioxide tension and pH values, is an important prognostic sign in infants with meconium aspiration and undergoing long-distance transfer for ECMO, whereas renal failure and intracerebral hemorrhage are usually lethal complications of ECMO. Each center performing ECMO should continually reevaluate this invasive technique and its results and complications.

(Ann Thorac Surg 1990;50:720–3)

Extracorporeal membrane oxygenation (ECMO) is becoming an accepted therapeutic option for infants and children with cardiorespiratory failure. Although this life support technique has been used for a variety of diseases, it has been most successful in the treatment of newborns with respiratory failure related primarily to reversible pulmonary hypertension. Numerous centers have reported their results with this technique, with the emphasis in the majority of these reports on patient selection, techniques of cannulation and perfusion, complications, and short-term follow-up of survivors. However, there is very little information available regarding the determinants of survival with this procedure. We critically analyzed a large number of newborns treated with ECMO to determine which factors are prognostic for survival, to better define the role of this invasive technique in the therapy of newborn respiratory failure and to modify selection criteria and management techniques if indicated.

Material and Methods

From January 1984 to December 1988, 135 newborns with respiratory failure unresponsive to conventional management were treated with ECMO. Conventional management varied because of a large number of referring neonatology units involved, but virtually always included trials of high-pressure, rapid-rate (100 to 150 breaths/min) ventilation with pharmacological support (inotropic agents, tolazoline). Occasional infants had received high-frequency or jet ventilation trials before ECMO as well. This group was selected from over 200 newborns who were referred for consideration for ECMO, some of whom were found not suitable because of previous selection criteria [1].

The technique of ECMO has been published previously [1, 2], and does not differ substantially from the methods originally described by Bartlett and associates [3]. Briefly, the carotid artery and jugular vein are exposed in the right side of the neck using local anesthesia. After patient heparinization (150 U/kg), each vessel is cannulated with an appropriate-sized polyethylene cannula, with the venous catheter advanced into the right atrium and the arterial catheter into the aortic arch. The membrane oxygenator, roller pump, heat exchanger, and reservoir are fashioned into a circuit by connections with polyethylene tubing, and the vessel cannulas are connected to the circuit. The venous blood drains by gravity into the reservoir, from which it is pumped through the oxygenator and heat exchanger. The rewarmed, oxygenated blood is returned to the baby via the carotid artery–aorta cannula. Systemic heparinization is continued by heparin drip adjusted to keep the activated clotting time at 200 to 250 seconds. Blood products, parenteral nutrition, antibiotics, and other intravenous medications are infused

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Table 1. Variables Examined for 135 Newborns Treated With Extracorporeal Membrane Oxygenation[a]

						Before ECMO		
Diagnosis	No.	Gest. Age (wk)	Birth Weight	Age on ECMO (h)	pH	Pco_2 (mm Hg)	Po_2 (mm Hg)	Time on ECMO (h)
CDH	25	38.7 ± 0.5	3.09 ± 0.1	36.5 ± 8.2	7.28 ± 0.03	50.0 ± 3.6	37.3 ± 4.2	118 ± 16.5
MAS	45	40.3 ± 0.2	3.5 ± 0.07	50.5 ± 8.8	7.35 ± 0.03	49.5 ± 5.6	35.4 ± 2.1	104.2 ± 10.3
PFC	42	38.0 ± 0.4	3.1 ± 0.1	69.6 ± 10.9	7.40 ± 0.03	36.2 ± 2.6	44.1 ± 3.3	136.4 ± 10.1
RDS	17	35.8 ± 0.9	2.45 ± 0.15	48.6 ± 10.3	7.32 ± 0.06	46.3 ± 6.0	36.8 ± 5.7	133.0 ± 21.1
Sepsis	6	36.7 ± 0.9	2.6 ± 0.7	38.2 ± 11.0	7.23 ± 0.1	46.3 ± 8.1	35.3 ± 5.6	137.8 ± 46.1
Mean (Group)		38.6 ± 0.9	3.2 ± 0.06	52.6 ± 4.9	7.34 ± 0.02	45.1 ± 2.3	38.4 ± 1.6	121.9 ± 6.3

[a] Values are shown as mean ± standard error of the mean.

CDH = congenital diaphragmatic hernia; ECMO = extracorporeal membrane oxygenation; Gest. = gestational; MAS = meconium aspiration syndrome; Pco_2 = carbon dioxide tension; PFC = persistent fetal circulation; Po_2 = oxygen tension; RDS = respiratory distress syndrome.

directly into the circuit. The flow rate is initiated at 100 to 150 mL · kg^{-1} · min^{-1} and adjusted to maintain the patient's oxygen tension at 80 to 100 mm Hg. While the patient is on ECMO, the ventilator is set to minimal values (rate, 10 breaths/min; peak inspiratory pressure, 15 cm H_2O; inspired oxygen fraction, 0.30).

As the patient's pulmonary function improves, the flow rate is decreased and the ventilator support is increased in increments. When the flow rate reaches 80 mL/min, a 10-minute trial-off period is attempted. If the blood gases are adequate, then ECMO is terminated, the cannulas are removed, and conventional ventilatory management is resumed.

Using these techniques, a total of 135 newborns were treated. The characteristics of the entire group are shown in Table 1. Because of the diversity of diagnoses and prognoses, we chose to analyze each diagnostic group individually for determinants of survival. The variables examined for each group included birth weight, gestational age, age at initiation of ECMO, best pH, oxygen tension, carbon dioxide tension before ECMO, and hours on ECMO. In addition, the impact of the two most serious complications of ECMO (renal failure and intracerebral hemorrhage) were analyzed with regard to the survival in the entire group. Because one of our selection criteria is a negative head ultrasound before the institution of ECMO, the intracerebral hemorrhages all developed after the infants had been placed on ECMO. Finally, a group of 50 infants who had undergone air transport for ECMO at our center were analyzed for the same criteria. It should be noted that infants with a diagnosis of "sepsis" all had blood cultures positive for *Streptococcus* sp and had respiratory failure either on the basis of pulmonary hypertension or adult respiratory distress syndrome equivalent.

Statistical analyses were completed using an Apple Macintosh II computer with statistical software (Statview) to test χ^2, analysis of variance, Student's *t* test, and multivariate analyses, where appropriate.

Results

The overall survival for this group of 135 infants treated with ECMO was 69%. Previous studies have determined that expected survival in this group is less than 10% [1, 3]. The individual survival rates for each diagnosis were: congenital diaphragmatic hernia, 56%; meconium aspiration syndrome, 87%; persistent fetal circulation, 69%; respiratory distress syndrome, 59%; and sepsis, 17%. Table 1 outlines the variables analyzed in the present series for the total population of newborns in each diagnostic group. Previous selection criteria [1] of term or near-term for candidates for ECMO explain the gestational age and birth weights. Examination of the blood gas data shows how critically ill these infants were before ECMO, with oxygen tension values in the 34 to 45 mm Hg range. The babies with a diagnosis of sepsis, respiratory distress, and diaphragmatic hernia required ECMO earlier than infants with the other diagnoses.

Table 2 compares survivors and nonsurvivors with regard to the same variables. There were no statistically significant differences that would predict survival in the infants with diaphragmatic hernia and sepsis. In two other groups, persistent fetal circulation and respiratory distress syndrome, only a single variable was found to distinguish survivors from nonsurvivors. In infants with persistent fetal circulation, the nonsurvivors had significantly more hours on ECMO when compared with survivors, a finding not unexpected as the nonsurvivors obviously had more severe pulmonary dysfunction. Infants with respiratory distress syndrome who did not survive ECMO were placed on ECMO significantly earlier than survivors, once again indicating a more severe pulmonary dysfunction that did not respond to conventional treatment.

In infants with meconium aspiration there was a clear difference in blood gas determinations before ECMO, with survivors having more nearly normal values than nonsurvivors, although the blood gas levels were not normal for either population. The carbon dioxide tension was particularly striking in differentiating survivors from nonsurvivors, indicating that the ability to ventilate, rather than oxygenate, is an important prognostic factor in infants with this diagnosis.

The patients who underwent long-distance air transfer to our facility for ECMO were further analyzed (Table 3) to attempt to predict which infants might be most able to tolerate this extremely dangerous and stressful maneuver.

Table 2. Analysis of Effect of Variables on Survival in Extracorporeal Membrane Oxygenation in Newborns[a]

Diagnosis		Gest. Age (wk)	Birth Weight (kg)	Age on ECMO (h)	pH	Before ECMO		Time on ECMO (h)
						Pco_2 (mm Hg)	Po_2 (mm Hg)	
CDH	Survivor							
	Nonsurvivor							
	p value	NS	NS	NS	NS	NS	NS	NS
MAS	Survivor				7.38 ± 0.03	43.7 ± 3.7	36.9 ± 2.2	
	Nonsurvivor				7.20 ± 0.13	89.8 ± 3.1	25.2 ± 5.7	
	p value	NS	NS	NS	0.01	0.003	0.05	NS
PFC	Survivor							123.0 ± 8.0
	Nonsurvivor							165.4 ± 25.6
	p value	NS	NS	NS	NS	NS	NS	0.04
RDS	Survivor			62.0 ± 10.6				
	Nonsurvivor			29.5 ± 10.1				
	p value	NS	NS	0.02	NS	NS	NS	NS
Sepsis	Survivor							
	Nonsurvivor							
	p value	NS	NS	NS	NS	NS	NS	NS

[a] Values are shown as mean ± standard error of the mean.

NS = not significant; all other abbreviations are as in Table 1.

It was very clear that the babies with a higher birth weight, more normal gestational age, and more normal pH and carbon dioxide tension levels were most likely to ultimately survive ECMO after air transport. Interestingly, oxygen tension levels were not predictive of survival in this group.

The correlation between renal failure and intracerebral hemorrhage during ECMO, and survival, was examined. Forty-three of the 135 patients (32%) in this series sustained renal failure (serum creatinine level greater than 180 μmol/L [2.0 mg/dL], and/or requirement for dialysis or ultrafiltration during ECMO because of decreased urine output). The patients with renal failure were distributed equally among all diagnoses. Eight of the 43 patients survived and 35 died, a highly significant difference ($p < 0.0001$). Likewise, there was a highly significant ($p < 0.001$) difference in survival with the development of intracerebral hemorrhage (25 patients total, 6 survivors), detected by daily cranial ultrasound during ECMO. Once again, the infants in whom intracerebral hemorrhage developed were distributed equally among all diagnoses.

Comment

Since the first description of the use of ECMO for newborn respiratory failure by Bartlett and associates in 1976 [3], the use of this technique has grown dramatically. It is now an accepted therapeutic modality for infants who are unresponsive to conventional therapy and a recent report from the ECMO National Registry shows over 1,800 survivors (personal communication, Dr Robert Bartlett, 1989).

Initial published reports have emphasized the techniques involved in the operative and patient management phases, the patient selection process, complications involved, and short-term follow-up of the initial patients. These topics have been well discussed in other recent reports, including one from this institution [4–6]. How-

Table 3. Effects of Variables on Survival After Long-Distance (Air) Transportation[a]

Outcome	No.	Gest. Age (wk)	Birth Weight (kg)	Age on ECMO (h)	pH	Before ECMO		Time on ECMO (h)
						Pco_2 (mm Hg)	Po_2 (mm Hg)	
Survival	28	38.9 ± 0.4	3.25 ± 0.11	76.6 ± 12.1	7.40 ± 0.04	38.5 ± 3.4	42.5 ± 3.4	116.9 ± 10.6
Nonsurvival	22	37.0 ± 0.7	2.74 ± 0.14	45.4 ± 7.1	7.24 ± 0.05	60.5 ± 9.1	33.5 ± 4.9	150.5 ± 22.3
p Value		0.02	0.005	0.04	0.005	0.02	0.13	0.15

[a] Values are shown as mean ± standard error of the mean.

Abbreviations are as in Table 1.

ever, a critical analysis of a large series from a single institution, using survival as the end-point, is important to further define the indications and candidates for the procedure and to develop exclusionary criteria if ECMO will not prove efficacious.

The present study demonstrates several important factors. Previously determined criteria [1] were used to select infants who were mature enough to tolerate such an invasive procedure (gestational age, 34 to 40 weeks; weight, greater than 1,800 g). The present analysis confirms the validity of these ranges, as the survivors and nonsurvivors in each diagnostic category were evenly distributed within these ranges. Thus the nonsurvivors died of causes other than those related to birth weight or gestational age. In addition, virtually none of the variables examined for each diagnostic category accurately predicted survival with the exception of the group of infants with meconium aspiration, among whom extremely low pH and oxygen tension values, combined with a high carbon dioxide tension, were associated with a poor prognosis. The explanation for such specificity to meconium aspiration is not clear. At present we are unable to exclude candidates for ECMO based on the criteria examined herein.

The selection criteria for which infants might tolerate long-distance transfer to an ECMO center for treatment are more clear from the analysis. The small infants (gestational age, less than 37 weeks; weight, less than 2.8 kg) who have an extremely low pH (primarily due to a high carbon dioxide tension) are very unlikely to survive, even if they survive the trip and are placed on ECMO. These findings must be kept in mind before undertaking an expensive, risky trip that might offer only false hope to distraught parents.

Two other variables were examined in this study to assess their effect on survival. The most serious complications that develop during and after ECMO are intracerebral hemorrhage and renal failure. Previous publications have emphasized the lethality of these complications [4, 7, 8], and this is confirmed in the present study. Although we have a few survivors with these problems, in general when either or both of these develop, survival becomes unlikely and the parents are so informed. When grade IV intracerebral hemorrhage develops on ECMO, as judged by daily cranial ultrasound, then discussion is held with the parents regarding the advisability of continuing the procedure. Our earlier experience suggests that these patients will invariably be neurologically devastated if they survive [7].

The complication of renal failure is certainly treatable with hemodialysis or ultrafiltration during ECMO [8]. However, renal failure in these patients is usually an indication of multiorgan failure, developing in many instances after the seventh day of ECMO. Renal failure is also a poor prognostic sign after cardiopulmonary bypass for cardiac operations, and thus it is not surprising to find it such an important prognostic determinant during ECMO.

Based on these data, we have modified our criteria for selecting infants for long-distance transfers to our facility for ECMO. We also believe our other criteria for selecting ECMO patients are valid and we will continue to use them. Each center performing ECMO should constantly analyze their results to further refine the indications for this life-saving technique.

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Acquired Benign Esophagorespiratory Fistula: Report of 16 Consecutive Cases

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Sixteen cases of acquired benign esophagorespiratory fistula were treated in a 20-year period. A delay in diagnosis was usual, and most patients were first seen with a pulmonary infection already developed. Contrast esophageal x-ray studies established the diagnosis in all patients. There were seven esophagotracheal and nine esophagobronchial fistulas. A fistula between the esophageal diverticulum and a bronchus considered to be of inflammatory origin developed in 7 patients. A fistula as the consequence of trauma developed in 9 patients, and these fistulas were situated at a higher level of the respiratory tree. All patients underwent surgical treatment; in 12 it was definitive, and in 4 temporary gastrostomy was performed to improve nutrition before definite repair. The definitive repair consisted of eventual diverticulectomy, division of the fistula, and suture of both esophageal and respiratory defects. Two patients required esophageal resection and later reconstruction with colon interposition. One patient died, creating an operative mortality of 8.3% in the definitive-repair group. The remaining 11 patients had a gratifying long-term result. There were two deaths in the gastrostomy group due to an extremely poor condition of patients and debilitating pulmonary infection. Early diagnosis of this rare condition is necessary if severe pulmonary complications are to be avoided. Early direct repair gives excellent results.

(Ann Thorac Surg 1990;50:724–7)

Benign esophagorespiratory fistula (BERF) is a rare condition and an unusual cause of chronic pulmonary suppuration in adults. Although the ratio of benign to malignant esophagorespiratory fistulas is around 1:5 [1], BERFs are of greater surgical interest because of their potential for total curability. Benign esophagorespiratory fistulas may be either congenital or acquired, with the latter being more common. Acquired BERFs are most commonly inflammatory or posttraumatic in origin, and an increasing number of the latter seem to have been recorded as a complication of blunt trauma to the chest in traffic accidents and secondary to cuffed endotracheal or tracheostomy tubes in patients requiring prolonged intubation and mechanical ventilation [2, 3]. The nonspecific nature of presenting symptoms makes the diagnosis sometimes difficult; the main classic finding of sudden coughing seconds after ingestion of fluids or solids (Ono's sign) should arouse suspicion of the diagnosis. Early diagnosis of BERF is necessary if severe pulmonary complications are to be avoided; this can almost always be established by contrast esophageal x-ray studies.

This report reviews the presentation and management of 16 consecutive cases of acquired BERF. As nonoperative management is generally unsuccessful and often fatal [1, 3, 4], the emphasis is on diagnosis and early surgical management.

Material and Methods

Patients

Sixteen patients with BERFs have been treated at the Institute for Digestive Diseases, Belgrade University Clinical Center, over a 20-year period ending December 31, 1988. The overall mean age of patients was 52.5 years (range, 25 to 68 years). The duration of symptoms before diagnosis was established ranged from 2 weeks to 6 years, and they were mostly of a chronic pulmonary nature. Paroxysmal coughing after fluid ingestion (Ono's sign) was present in 13 patients (81%). Hemoptysis was a symptom in only 1 patient, but it was not severe. Fever and recurrent pulmonary infections were the predominant symptoms in 2 patients (12.5%). Associated pulmonary disease was present in 12 patients (75%) and included pneumonia in 9, bronchiectasis in 2, and empyema in 1. In all patients, the diagnosis was established by means of contrast radiography. This demonstrated esophagotracheal communication in 7 patients and esophagobronchial communication in 9. Esophagoscopy and bronchoscopy indicated the site and extent of fistula in 11 patients (69%). Biopsy specimens and pulmonary secretions were retrieved for culture at the time of endoscopy. There were nine posttraumatic fistulas in this series; their site, probable duration of fistula (interval between trauma and diagnosis), and mechanism of injury are shown in Table 1. The mean age of these 9 patients was 47.5 years (range, 25 to 68 years).

Diverticulorespiratory communication was demonstrated in 7 subjects, and these fistulas were considered to be of inflammatory origin. All of these were on the right side of the respiratory tree: two with the right main

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Table 1. Site, Interval to Symptoms and Diagnosis, and Mechanism of Injury in 9 Patients With Posttraumatic Benign Esophagorespiratory Fistula

Patient No.	Site	Interval		Mechanism of Injury
		T/S	T/D	
1	Esophagotracheal (upper third)	1 wk	4 mo	Tracheostomy tube; NG tube?
2	Esophagotracheal (upper third)	4 d	3 wk	Tracheostomy tube; NG tube?
3	Esophagotracheal (upper third)	2 wk	10 mo	Tracheostomy tube
4	Esophagotracheal (middle third)	14 mo	7 y	Blunt chest trauma (traffic accident)
5	Esophagotracheal (lower third)	2 mo	2 mo	Blunt chest trauma (traffic accident)
6	Esophagotracheal (lower third)	1 mo	3 mo	Ingestion of dental prothesis, impaction, endoscopic removal
7	Esophagotracheal (lower third)	4 wk	6 wk	Blunt chest trauma (traffic accident)
8	Esophagobronchial (left main)	5 mo	5 mo	Chemical injury (lye ingestion)
9	Esophagobronchial (right main)	5 d	1 y	Blunt chest trauma (fall from height)

NG = nasogastric; T/D = trauma/diagnosis; T/S = trauma/symptom(s).

bronchus and five with a secondary or tertiary bronchus. *Mycobacterium tuberculosis* was identified as a possible causative organism in only 1 patient. The mean age of these 7 patients was 62 years (range, 58 to 68 years) and the late presentation of their symptoms would favor the acquired origin of lesions.

Methods

All patients underwent surgical treatment, 12 of them definitive; in the remaining 4 a feeding Stamm gastrostomy was performed as a primary procedure to improve the nutritional status before definitive repair. Patients scheduled for definitive repair underwent preoperative antibiotic therapy and correction of malnutrition for at least 10 days. The procedures performed were direct repair in 5 patients, esophageal diverticulectomy and closure of tracheal/bronchial defect in 5, esophagectomy, closure of bronchial defect and substernal colon interposition in 2, and feeding gastrostomy in 4. Direct repair was achieved via a right posterolateral thoracotomy, with division of the fistula and closure of the fistulous tracts into both the esophagus and respiratory tract. A pleural flap was interposed between the esophageal and tracheal/bronchial suture lines where possible. In all patients, the esophagus was closed in two layers of interrupted, nonabsorbable sutures. Associated esophageal diverticula were resected, with closure of the esophageal and respiratory defects in the same manner. In 2 patients, esophagectomy was performed owing to a scarred and strictured esophagus, and the respiratory defects were sutured and pleuralized; the continuity of the digestive tract was restored by substernal coloplasty on the left colic artery in the manner previously described by Gerzić and associates [5].

Stamm gastrostomy was carried out only when the nutritional status and debilitating pulmonary infection precluded a major operation. One of these patients underwent endoscopic cauterization of the fistula twice, but without any improvement.

Operative mortality was defined as death within 30 days of operation or directly related to it. Follow-up for survivors ranged from 1 to 6 years (mean, 2.8 years) and consisted of a clinical interview, barium swallow, and endoscopy. Patients were considered to have excellent results when practically asymptomatic.

Results

Eleven of the 12 patients who underwent definitive surgical repair had an unremarkable postoperative course and an excellent long-term result (Fig 1). There were no recurrent fistulas up to 6 years postoperatively and none of the patients suffered recurrent pulmonary infections.

One patient, aged 68 years, who underwent diverticulectomy and the closure of a bronchial defect died 36 days after the operation with the signs of bilateral pneumonia and myocardial failure, creating a postoperative mortality of 8.3% in the group that underwent definitive surgical correction. The integrity of the esophageal suture line in this patient was confirmed by a Gastrografin swallow on the eighth postoperative day, and the patient received regular peroral food requirements until the time of death.

There were two deaths in the gastrostomy group (group mortality, 50%) caused by an extremely poor general condition of patients, including debilitating pulmonary suppuration and cachexia. Both patients had an undetected lesion for a period of 1 and 2 years after trauma and an absent Ono's sign. Considered to have cases of advanced

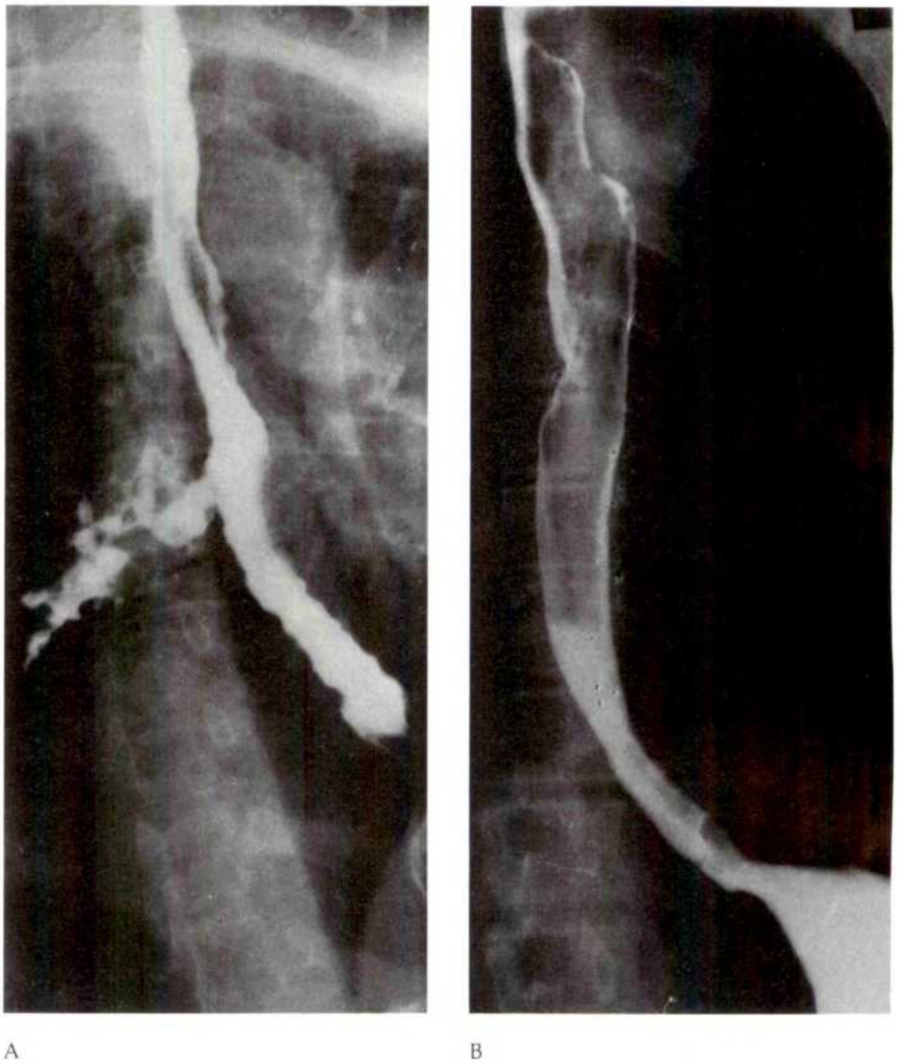

Fig 1. (A) Barium swallow demonstrates diverticulobronchial fistula. (B) Esophagram after repair consisting of diverticulectomy and closure of bronchial defect.

malignant cachexia due to esophageal cancer, they were sent for operation when 1 of them already had well-developed empyema of the pleura.

Of the remaining 2 patients from this group, 1 refused any further treatment and is lost to follow-up; the other gained some weight, has no signs of further pneumonia, and is planned for definitive repair soon.

Three deaths created the overall postoperative mortality of 18.7%.

Comment

Acquired BERFs are rarely seen and are most commonly the consequence of trauma or of an inflamed mid-thoracic traction diverticula. The correct diagnosis can be elusive, especially when Ono's sign is absent. Most patients are first seen with chronic pulmonary suppuration as the predominant but late symptom. Traumatic esophagorespiratory fistula is usually a delayed diagnosis, principally due to the elapse of time before posttraumatic necrosis of the esophageal wall results in fistulation [6]. One of our patients in whom a correct diagnosis was established 6 years after the trauma stated that the first symptoms appeared 14 months after the event. Thorough evaluation for occult esophagorespiratory fistula should be a part of the examination of every patient with chronic pulmonary suppuration of obscure etiology if serious and potentially preventable complications are to be avoided and years of debilitating illness prevented.

The diagnosis can almost always be established by roentgenographic examination of the esophagus with contrast material; thorough examination of the esophagus provides information about the fistula site and size but does not give sufficient data about the nature of the lesion. Esophagoscopy and bronchoscopy were less helpful in our hands in establishing the diagnosis and failed to reveal a fistula in nearly one third of patients. We found both methods of more use in excluding malignancy and for procurement of material for bacteriological and histopathological analysis.

A clear distinction has been made between posttraumatic and inflammatory fistulas according to the site of the lesion. Although all inflammatory fistulas were beneath the bifurcation of the trachea on the right side of the respiratory tree, posttraumatic fistulas were situated at a higher level. In 7 patients they were associated with the trachea and in 2 with the main bronchus (one with the right and one with the left).

In all 3 patients in whom a fistula developed after tracheostomy, the lesion occurred at the level of the inflated cuff. Two of these patients had a simultaneous nasogastric tube, which probably contributed to the pressure necrosis [3].

Two of 4 patients in whom a fistula developed from nonpenetrating thoracic trauma experienced the first symptoms 4 and 9 weeks after the injury with the signs of mediastinal infection having already developed. This suggests the possibility of an inflammatory origin for these fistulas. Although differentiation is practically impossible, it is clear that at least some of these fistulas are related to a posttraumatic infectious process. A similar situation was observed in the only case of left esophagobronchial fistula, which occurred 5 months after the ingestion of sodium hydroxide for suicide attempt.

The remaining 2 patients in whom a fistula developed from nonpenetrating thoracic trauma did not have any bone fractures of the chest wall. Trauma probably resulted from compression of trachea and esophagus between the sternum and vertebrae at the time of impact [7].

Although spontaneous healing of acquired esophagorespiratory fistulas has been reported [1], direct surgical repair proved to be the procedure of choice and yields gratifying results [4, 8]. As most patients are seen with an advanced pulmonary infection, diligent preoperative preparation of these patients is necessary, along with the use of appropriate antibiotics when indicated and respiratory exercises.

We found a right posterolateral thoracotomy to be an excellent approach for these patients because it provides exposure of the left main bronchus and lower two thirds of the trachea as well. In 1 case of esophagotracheal fistula after tracheostomy, the initial attempt at repair was carried out through a cervical approach but adequate exposure was not obtained and a transthoracic approach had to be used. The cervical approach should be reserved only for those fistulas occurring well above the clavicula, but these are very rare and we have not seen any of them in this series.

An integral part of the direct-repair technique requires that all adjacent mediastinal nodes and necrotic tissue be excised. As diverticulorespiratory communications were caused by the traction diverticula, we did not find myotomy necessary in these patients; none of them experienced any swallowing problem up to 6 years postoperatively.

Occasionally, the severely damaged esophagus may preclude direct repair and may even become an indication for esophagectomy. In 2 such cases, continuity of the digestive system was restored by left colon interposition. The operation can be carried out either as a one-stage or a two-stage procedure, depending on the presence of mediastinal infection. If present, we consider esophagectomy, suture of the respiratory defect, temporary diverting esophagostomy, and feeding gastrostomy to be safer as the initial procedure, whereas colon interposition should be carried out when infection is controlled at a later date.

The high overall postoperative mortality reported in this series was principally the consequence of a delayed diagnosis, advanced pulmonary suppuration, and the extremely poor medical condition of the patients. If this high mortality is to be avoided, increased awareness of this rare but debilitating condition is needed on the part of all physicians dealing with respiratory disease [9].

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Correction of Truncus Arteriosus With Truncal Valvar Stenosis or Insufficiency Using Two Homografts

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Surgical correction of truncus arteriosus requires the creation of right ventricular to pulmonary artery continuity and closure of the ventricular septal defect. A variety of conduits have been used including valved and nonvalved. Despite a significant incidence of truncal valvar stenosis and insufficiency, this valve has seldom been replaced. We present 4 cases of truncus arteriosus with truncal valvar stenosis or insufficiency that were repaired using two valved homografts: one to create the pulmonary outflow tract and the other to replace the abnormal truncal valve. Two of these patients are doing well after 4 months. Another child survived the operation and did well for 2 months when she died suddenly. The last child died 14 hours postoperatively from low cardiac output syndrome secondary to diabetic hypertrophic cardiomyopathy. When truncal valvar abnormalities are present, the primary repair of truncus arteriosus in an infant should include replacement of the truncal valve. Total correction can be successfully achieved using two valved homografts, resulting in long-term palliation and freedom from thromboembolic events and the use of anticoagulants.

(Ann Thorac Surg 1990;50:728–33)

Truncus arteriosus is a rare congenital heart malformation that results from failure of septation of the embryonic truncus arteriosus as well as failure of normal septation of the conus. A single arterial trunk arises from the base of both ventricles through which the entire circulation passes. This defect was first successfully repaired in 1962 using a nonvalved plastic conduit [1] and then in 1967 by McGoon and associates [2] using a valved aortic homograft to construct a pulmonary outflow tract. Subsequently, either a Dacron graft with a porcine valve [3] or a valveless prosthetic conduit [4] was the preferred means of creating right ventricle to pulmonary artery continuity. Recently, Bove and co-workers [5] reported excellent results using aortic or pulmonary homografts as the pulmonary conduit and replacing insufficient and stenotic truncal valves with a disc prosthesis. Regardless of the repair method, controversy continues over the proper age at which to perform the initial repair and how to manage truncal valve regurgitation and stenosis [6].

As our experience with homograft conduits has increased [7], we have corrected truncus arteriosus in 4 infants with truncal valve abnormalities by using two separate homograft conduits. One was used to create the right ventricular outflow tract, and the other was used to create the left ventricular outflow tract, concurrently replacing the abnormal truncal valve. This has resulted in excellent functional results, avoiding residual problems secondary to the presence of an abnormal truncal valve.

Material and Methods

From October 9, 1986, to May 25, 1989, 4 infants with truncus arteriosus and an incompetent or stenotic truncal valve underwent surgical correction at Children's Hospital of Oklahoma by using two valved homograft conduits. One child died shortly postoperatively secondary to diabetic hypertrophic cardiomyopathy. Three survived and at follow-up had excellent functional results, which were demonstrated clinically and by echocardiography. Of these 3 survivors there has been one late death, the cause of which was probably noncardiac.

Patient 1

A female, 2.4-kg, term infant, the product of a normal pregnancy, was delivered on July 12, 1986. She was found to have a type II truncus arteriosus with a quadricuspid valve that had both mild stenosis (16 to 20 mm Hg) and insufficiency. Mild congestive heart failure was treated successfully with digoxin and furosemide. On October 1, 1986, sudden exacerbation of the congestive heart failure developed necessitating intubation and inotropic support. On October 9, 1986, total correction was performed using two aortic homografts to replace the truncal valve and to create a pulmonary conduit. The ventricular septal defect was closed with the anterior leaflet of the mitral valve of the homograft in the aortic position. Postoperatively, moderate inotropic support for 6 days and vigorous diuresis were required. Isoproterenol hydrochloride (Isuprel) was administered briefly for intermittent bradycardia. In addition, an episode of *Enterobacter* and *Acetobacter* line sepsis necessitated a 7-day course of tobramycin.

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At discharge on postoperative day 16, the child was taking only digoxin. She was eating well and had made an appropriate weight gain.

Follow-up at 3 weeks found a healthy, active, 3.4-kg infant. Echocardiography revealed functioning homografts, very mild aortic and pulmonic insufficiency, and improved ventricular function. No ventricular ectopy was noted. The child continued to do very well until she was found dead in her crib on the morning of December 3, 1986, by her parents. Autopsy revealed a normal heart with both homografts appropriately attached. No inflammatory cell infiltrates or ischemic areas were seen microscopically. However, mild pulmonary edema was noted and *Staphylococcus aureas* was isolated in her blood.

Patient 2

A male, 4.7-kg, term infant, the product of a pregnancy complicated by maternal diabetes and polyhydramnios, was born vaginally on March 15, 1989. Echocardiography revealed type II truncus arteriosus with a quadricuspid valve that had both moderate stenosis (35 to 40 mm Hg) and insufficiency. Very severe asymmetrical biventricular hypertrophy and a small atrial septal defect were also noted. Clinically significant congestive heart failure, respiratory distress, and hypoglycemia were treated aggressively with inotropic drugs, diuretics, mechanical ventilation, and glucose. On March 30, 1989, total correction was achieved by using two aortic homografts to replace the truncal valve and to create a pulmonary valve and conduit. Because of the inadequate size of the mitral valve leaflet of the homograft in the pulmonary position, the ventricular septal defect was closed with a knitted Dacron patch. Weaning this child from extracorporeal bypass was difficult and required the use of inotropic agents.

Postoperatively the child had a persistent low cardiac output syndrome. Despite maximal inotropic support and resuscitative efforts, he died 14 hours after discontinuation of extracorporeal bypass. Autopsy revealed severe biventricular hypertrophy and cardiomegaly, consistent with diabetic hypertrophic cardiomyopathy.

Patient 3

A male, 3.7-kg, term infant, who was the product of a normal pregnancy, was delivered on March 5, 1989. He was found by echocardiography to have type I truncus arteriosus with a quadricuspid valve that had both mild stenosis (25 to 30 mm Hg) and moderate insufficiency. At 11 days of age congestive heart failure developed, and the child was treated with digoxin and furosemide (Lasix). On March 31, 1989, total correction was performed using two aortic homografts to replace the truncal valve and to create a pulmonary outflow tract. The anterior leaflet of the mitral valve of the homograft in the pulmonary position was employed to close the ventricular septal defect.

Postoperatively, moderate inotropic support for 4 days and vigorous diuresis were necessary. On the fifth postoperative day an echogenic mass thought to be thrombus causing mild insufficiency was identified on one aortic valve leaflet. Both the thrombus and insufficiency resolved with a 3-day infusion of streptokinase. Nonsustained ventricular ectopy noted in the postoperative period responded to propranolol.

At discharge on postoperative day 19, medications included propranolol, digoxin, and aspirin. The child was eating and gaining weight appropriately.

At follow-up at 4 months, the child was healthy and active, and weighed 6.6 kg. Administration of propranolol had been discontinued and no further ventricular ectopy was noted. Digoxin administration will be continued for approximately 2 more months. Echocardiography revealed improved ventricular contractility, a competent aortic valve, and mild pulmonary insufficiency.

Patient 4

A female, 3.2-kg, term infant, the product of a normal pregnancy, was born on May 6, 1989. Because of a heart murmur and episodes of cyanosis, an echocardiogram was obtained. This revealed a type II truncus arteriosus with a quadricuspid truncal valve that was both stenotic (52 mm Hg) and insufficient, and an atrial septal defect. On May 25, 1989, total correction was performed using two aortic homografts to replace the truncal valve and to create a pulmonary conduit. The anterior leaflet of the mitral valve of the homograft in the pulmonary position was used to close the ventricular septal defect. The atrial septal defect was closed through an atriotomy in the usual fashion.

Postoperatively the child required mild inotropic support for 6 days. She was eating well and in no distress until postoperative day 9 when respiratory distress developed and she was noted to have hepatomegaly. Administration of digoxin, furosemide, and spironolactone was started and her symptoms resolved.

At discharge on postoperative day 13, medications included digoxin, furosemide, and spironolactone. The child was eating, gaining weight, and active.

At follow-up 4 months later, the child was healthy and active and weighed 4.5 kg. Administration of spironolactone had been discontinued, but she continued to take digoxin and furosemide. An echocardiogram revealed mild pulmonary insufficiency and stenosis (<15 mm Hg) along with mild mitral and aortic insufficiency.

Operative Technique

Through a median sternotomy the truncal vessel and right atrium are cannulated and extracorporeal bypass is initiated. The truncal vessel cannula is placed distal to the origin of the pulmonary arteries. Both the right and left pulmonary arteries are occluded by tourniquets. The patient is cooled to 15°C, an aortic cross-clamp is applied, and circulatory arrest is instituted after administration of cardioplegia. The truncal vessel is opened through a vertical incision slightly distal to the truncal valve (Fig 1). This allows inspection of the truncal valve, the coronary anatomy, and the confluence of the pulmonary arteries. The truncal valve is then totally excised, and the pulmonary arteries are excised from the truncus with an elliptical incision. The left and right coronary arteries (if both are present) are mobilized and the truncal vessel is divided leaving a convenient length for anastomosis of the aortic conduit (Fig 2). Extreme caution in mobilization of the coronary arteries must be exercised as the coronary arterial anatomy in truncus arteriosus is variable [8].

The truncal valve is replaced with an appropriate-sized cryopreserved aortic homograft (usually a 10- to 12-mm homograft). The homograft is trimmed as in a root replacement and the anterior leaflet of the mitral valve is removed. The homograft is sutured to the annulus of the truncal valve with a running 5-0 Prolene (Ethicon, Somerville, NJ) suture along the margin of the patient's anterior mitral leaflet and continued to the edge of the ventricular septal defect (Fig 3). The homograft is inserted in its anatomical position. The coronary arteries are reimplanted to small buttonholes made in the base of the homograft with a running 7-0 Prolene suture. If the coronary anatomy is normal, the left coronary artery can be implanted into the left coronary sinus after removal of a button of aortic homograft containing the left coronary ostium. The right coronary artery can then be implanted at an appropriate site on the homograft being careful to avoid excessive tension or kinking of the right coronary artery. The distal anastomosis of the homograft and truncal vessel is performed using a running 5-0 Prolene suture. At this time a second dose of cardioplegia is administered.

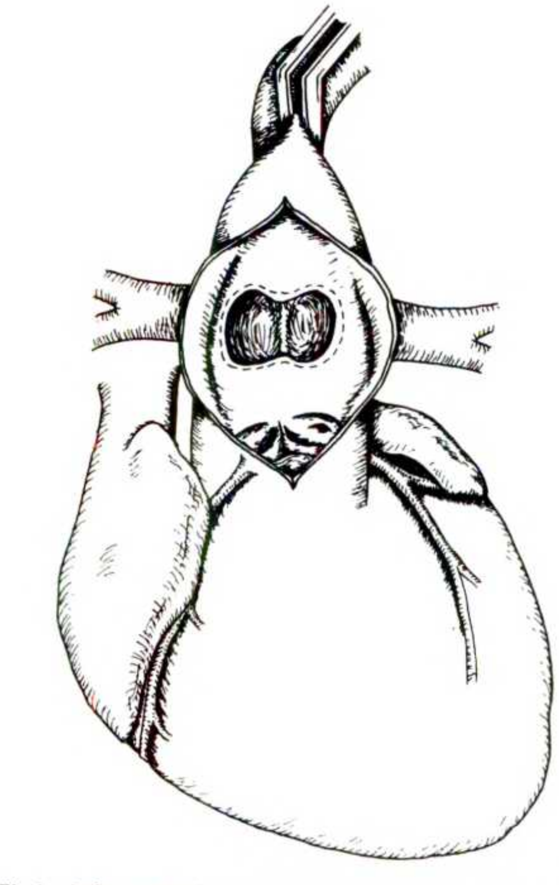
Fig 1. A short vertical arteriotomy allows inspection of the truncal valve, the coronary ostium, and the origin and confluence of the pulmonary arteries. Planned excision of the confluence of the pulmonary arteries is shown by the dotted line.

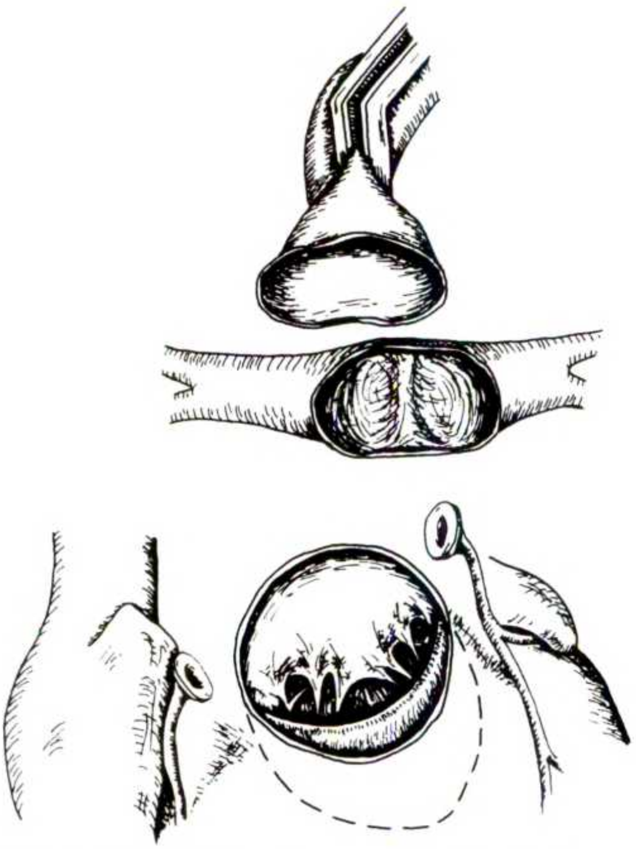
Fig 2. Mobilization of the coronary arteries with a cuff of truncal vessel, excision of the truncal valve and vessel, and the planned enlargement of the right ventricular outflow tract is shown by the slashed line.

A new pulmonary artery and valve are constructed using a second valved aortic homograft. The right ventricular outflow tract is opened widely by removing a portion of the muscular infundibulum (see Figs 2, 3). The pulmonary conduit is inserted by suturing the anterior leaflet of the mitral valve of the homograft to the ventricular septal defect using a running 5-0 Prolene suture buttressed with a Teflon felt pledget (Fig 4). This suture line continues to the right ventricle and completes the anastomosis of the new pulmonary artery to the right ventricle. The two homografts are then sutured at their point of apposition to close the left ventricular outflow tract with 5-0 Prolene suture.

The distal anastomosis of the conduit to the confluence of the patient's pulmonary arteries is accomplished as an end-to-end anastomosis (Fig 5). If present, an atrial septal defect is now closed in the usual fashion through a right atriotomy.

The patient is rewarmed, bypass is discontinued, anticoagulation is reversed, and hemostasis is meticulously obtained. Temporary pacing wires are placed on the right atrium and right ventricle. The pericardium is loosely closed and the chest is closed in a usual fashion.

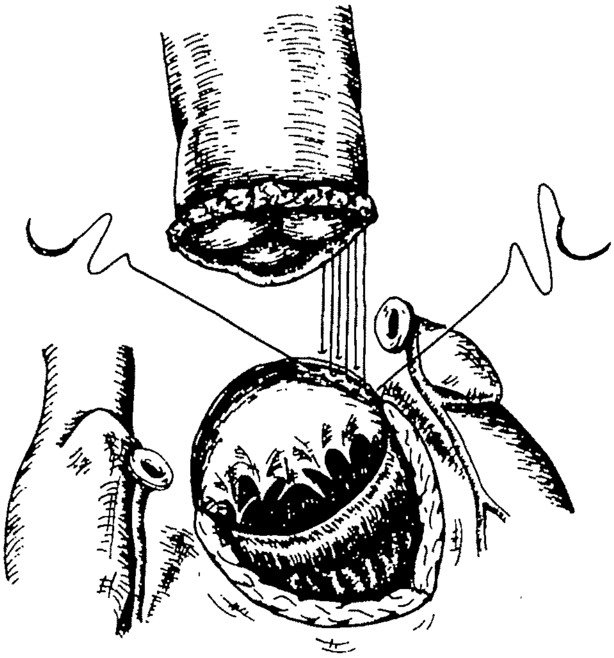

Fig 3. The aortic homograft used to replace the truncal valve is sutured to the truncal valve annulus beginning along the anterior leaflet of the mitral valve and continuing to the right ventricular margin of the ventricular septal defect.

Comment

Repair of truncus arteriosus is usually achieved by creating a right ventricle to pulmonary artery conduit. Valved homografts have been employed [2, 5], but most centers have used prosthetic grafts with or without porcine valves [1, 3, 4]. Bove and co-workers [5] recently reported a series of patients of which 2 had abnormal truncal valves replaced by a disc prosthesis in addition to creation of the pulmonary conduit with a pulmonary or aortic homograft. They suggested that replacing the diseased truncal valve with a valved aortic homograft may offer a substantial advantage over use of a mechanical valve.

The truncal valve consists of from one to six cusps. The majority are tricuspid (42%), followed by bicuspid (30%), quadricuspid (24%), and rarely, unicommissural (4%) [9]. Moderate to severe truncal valve insufficiency has been reported in 37% to 75% of the cases [10–12]. Microscopically the insufficient valves are noted to have irregular expansion and proliferation of the spongiosa and fibrosa layers [11, 12]. This results in thickened cusps of unequal sizes. In the absence of truncal valve insufficiency, there appears to be a significantly better response to digitalis and diuretics, and death may not be related to the cardiac lesion. Those with insufficiency do not respond as well to similar medical therapy and succumb to refractory congestive heart failure [11]. It has also been shown that, if treated surgically without valve replacement, patients with moderate to severe truncal valve insufficiency have a significantly reduced long-term survival rate [10].

Truncal valve stenosis, first reported by Burnell and associates [13], appears to be less prevalent than truncal valve insufficiency. The reported incidence of valvar stenosis has ranged from 5.6% to 11% [14, 15]. However, by using measurements of the valve orifice, of the valve ring and maximal diameter of the truncus, Gerlis and colleagues [16] calculated that stenotic truncal valves were present in 33% of their specimens. This same study revealed that the stenotic valves were more common in valves with two or four cusps, had a gradient of 13 to 40 mm Hg (mean, 21.5 mm Hg), and resulted from dysplastic cuspid thickening.

Presently more surgeons are using homografts for the repair of truncus arteriosus, citing the avoidance of a rigid prosthesis, a greater technical ease of suturing, and im-

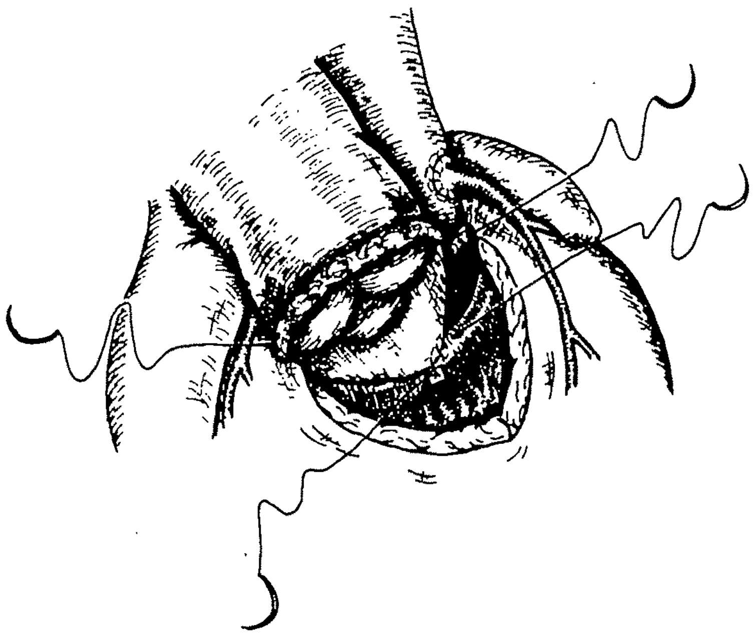

Fig 4. The aortic homograft for right ventricular outflow tract reconstruction has been trimmed and its anterior leaflet of the mitral valve is sutured to the margin of the ventricular septal defect on the right ventricular aspect to close the ventricular septal defect. The annulus of the aortic homograft is sutured to the margin of the right ventricle at the site of enlargement to complete reconstruction of the right ventricular outflow tract.

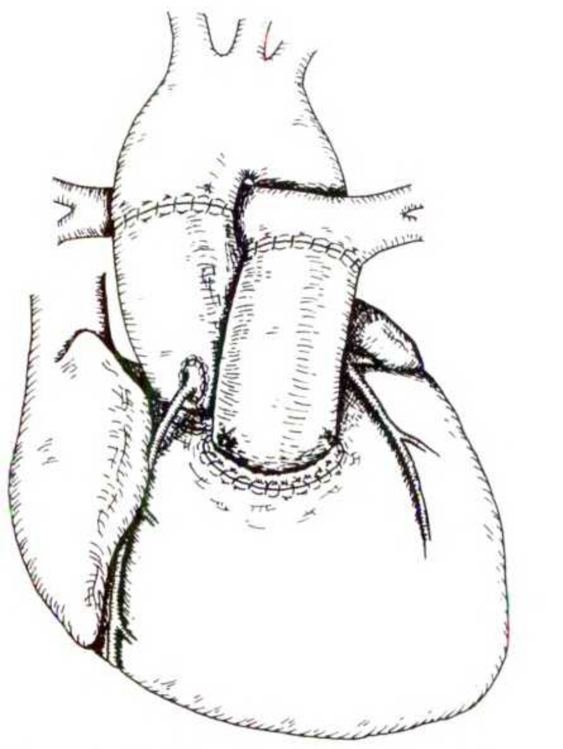

Fig 5. The completed operation shows replacement of the truncal valve with an aortic homograft, reimplantation of the coronary arteries, and reconstruction of a right ventricular outflow tract with an aortic homograft.

proved hemostasis [5]. Others state that the use of homografts allows the right ventricle to better compensate for acute episodes of pulmonary hypertension that sometimes occur in the early postoperative period [17]. The availability of homograft valves is increasing as both the medical and lay public become more educated about the needs for such conduits. Further, the use of such conduits is attractive in the neonate because of the availability of small homografts. Before the use of the homograft, the smallest conduit available was the 12-mm Dacron conduit containing a porcine valve, which was often technically difficult to insert in the neonate.

Improved techniques, including rapid homograft procurement, short period of antibiotic treatment, and prompt cryopreservation, appear to provide viable homografts with enhanced durability [18]. Cryopreservation has increased the availability of homografts and may lead to improved long-term success rate of the homograft valve conduit. Kay and Ross [19] reported that only three, 13% ± 8%, of their patients who had undergone right ventricular outflow tract reconstruction with aortic homografts required conduit replacement for obstruction after 10 years.

To avoid the long-term sequelae, Bove and co-workers have recommended that the truncal valve be replaced when severe regurgitation is present, especially in the presence of associated stenosis. Ebert and associates [20] also recommend replacement of the insufficient truncal valve, reporting that of the eleven operative deaths in their series, 8 patients had clinically significant truncal valve insufficiency. At our institution we concur with this premise. Because of the recent success reported with the use of the homografts in the pulmonary position, we have expanded this to include replacing the truncal valve with an aortic homograft. The early results have been excellent in 3 patients treated in this manner. The only operative death in our series was related not to the surgical procedure, but to the presence of diabetic hypertrophic cardiomyopathy. The surviving patients had excellent functional results with only echocardiographic evidence of mild valvar insufficiency and no obstruction. Three of the patients who survived the operation required digoxin and only 1 needed diuretic therapy. The 1 child who died suddenly 2 months postoperatively had no evidence of obstruction or valvar abnormalities at autopsy. Whether this late death was due to a cardiac arrhythmia or some other event is not known. Septicemia is suspected because of the positive cultures obtained at postmortem examination. The use of homograft conduits and valves in this type of repair results in excellent hemodynamic efficiency that may result in long-term palliation before growth necessitates reoperation. Even more importantly, using these conduits has the distinct advantage of freedom from disabling thromboembolic events and the requirement of anticoagulants, which have associated complications.

In conclusion, correction of truncus arteriosus in the neonate should include the replacement of the truncal valve when clinically significant insufficiency or stenosis is present. This undoubtedly will help to avoid long-term detrimental sequelae caused by the presence of an abnormal truncal valve. The use of fresh or cryopreserved aortic or pulmonary homografts offers an excellent means for correcting this lesion and avoids the inherent complications of the mechanical valves and Dacron conduits. A longer period of follow-up and a larger patient population will need to be evaluated, but our early results imply that this method of repair should be seriously considered when the cardiothoracic surgeon is faced with a child having truncus arteriosus with truncal valve stenosis or insufficiency.

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Notice to Readers of *The Annals of Thoracic Surgery*

Dr Richard M. Peters, who has functioned as an Associate Editor in charge of book reviews for the past 11 years, at his request retired from this position at the time of the Interim Meeting of The Society of Thoracic Surgeons in Chicago, September 21, 1990. He will remain as an Editorial Board member. Dr Peters has performed a great service for the readers of *The Annals*, and we are grateful to him.

Dr J. Kent Trinkle has agreed to be the new Associate Editor in charge of book reviews. Any correspondence concerning this section of *The Annals* should be sent directly to him at the address listed below:

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Influence of Prosthetic Design on Durability of the Liotta Porcine Valve in the Mitral Position

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From March 1979 to December 1984, the Liotta low-profile porcine bioprosthesis was employed for mitral valve replacement in 71 patients to avoid potential left ventricle–prosthesis mismatch occasionally observed with the standard, high-profile, Hancock porcine xenograft. Follow-up of 61 operative survivors showed at 10 years an actuarial survival of 67% ± 7%, freedom from thromboemboli of 96% ± 2%, freedom from structural deterioration of 63% ± 11% and freedom from all Liotta bioprosthesis-related complications of 53% ± 10%. Complications related to excessive protrusion of the stent into the left ventricular cavity were eliminated with the Liotta bioprosthesis; the peculiar stent configuration, however, was responsible for an increased rate of structural deterioration requiring reoperation in 10 patients (2.8% ± 0.9%/patient-year) at a mean interval of 76 ± 18 months after mitral valve replacement (range, 45 to 106 months). Common findings in all explants were cusp prolapse, cusp tears, and commissural rupture related to various degrees of tissue calcification, constantly leading to severe prosthetic incompetence. As also shown experimentally, such structural changes have been attributed to increased systolic stresses on the closed cusps, favored by excessive reduction of the stent height. Our experience shows that the Liotta bioprosthesis used for mitral valve replacement does not provide any clear-cut advantage over standard porcine bioprostheses and that its long-term durability appears affected by the unique prosthetic design.

(Ann Thorac Surg 1990;50:734–8)

The Liotta bioprosthesis (LPB) is a glutaraldehyde-treated porcine xenograft that, owing to a unique mounting technique and stent configuration, has a considerably lower profile when compared with other bioprostheses [1]. The LPB has been used clinically since 1976 [1, 2] mainly to eliminate complications caused by excessive protrusion of the stent into the left ventricle observed after mitral valve replacement (MVR) using standard porcine bioprostheses [3, 4].

We have reviewed the long-term follow-up of patients who underwent MVR with an LPB to assess the clinical performance of this device; our experience shows that long-term durability of the LPB appears to be affected by its particular prosthetic design.

Material and Methods

From March 1979 to December 1984, 71 patients underwent MVR using an LPB as an alternative to the Hancock porcine valve, which has been the bioprosthesis of choice at our department in the last 15 years [5]. The LPB was employed almost exclusively in the mitral position, usually in patients with a small left ventricular cavity, ie, those with pure or predominant mitral stenosis.

There were 53 female and 18 male patients, with a mean age at operation of 53 ± 12 years (range, 22 to 69 years). Isolated MVR was performed in 56 patients, whereas in 15 MVR was associated with replacement of the aortic valve using different prostheses (Hancock porcine in 7, Carpentier-Edwards porcine in 3, Hancock pericardial in 3, Sorin tilting-disc in 1, and Lillehei-Kaster tilting-disc in 1); in 6 patients with isolated MVR a tricuspid annuloplasty was also performed. Preoperatively 8 patients were in New York Heart Association class II, 51 were in class III, and 12 were in class IV.

All operations were performed with standard, moderately hypothermic cardiopulmonary bypass, topical cooling, and cold potassium cardioplegia. Liotta bioprostheses were inserted using multiple nonabsorbable sutures, usually reinforced by small pieces of Teflon felt.

Management of postoperative anticoagulation was similar to that adopted for recipients of other bioprostheses [5, 6]. Anticoagulation was suspended 3 to 6 months after operation except in patients with a large, fibrillating left atrium, those with left atrial thrombosis, and those receiving concomitantly a mechanical prosthesis.

Follow-up study, conducted during a 5-month period ending in May 1989, included direct patient reevaluation and analysis of questionnaires completed by the patient or the referring physician. Criteria used for definition of major postoperative complications were adopted following recently suggested guidelines [7]. Incidence of such

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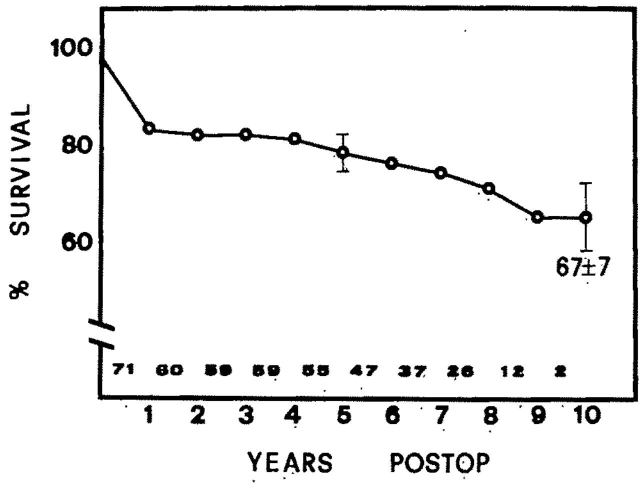

Fig 1. Actuarial survival after mitral valve replacement with the Liotta low-profile porcine bioprosthesis. Numbers on the horizontal axis indicate patients at risk. Operative deaths are included.

complications was calculated in both actuarial and linearized fashion and expressed as percent and percent per patient-year (%/pt-y), respectively. All actuarial curves were drawn including operative mortality.

Results

Early and Late Mortality

There were 10 operative deaths with an overall hospital (<30 days) mortality of 14% (70% confidence limits, 9.7% to 19.6%). Two patients died in the first postoperative hours because of left midventricular rupture; 6 died of low output syndrome, 1 of septic shock, and 1 of liver failure at various intervals after operation.

Follow-up of the 61 operative survivors ranged from 0.1 to 10.8 years (mean, 5.8 ± 3 years) with a cumulative duration of 358 patient-years, being 100% complete. During the follow-up period there were nine late deaths (2.5 ± 0.8%/pt-y); the cause of death was cardiac failure in 4 patients, unknown in 4, and a cerebral embolism in 1. Actuarial survival at 8 and 10 years is 76% ± 6% and 67% ± 7% (Fig 1). Out of 42 current survivors 5 are in New York Heart Association class I, 31 in class II, 5 in class III, and 1 in class IV.

Postoperative Complications

Two patients sustained major thromboembolic accidents (0.6 ± 0.4%/pt-y): in 1, peripheral embolization occurred 4 years after MVR requiring embolectomy, whereas the other died as a result of cerebral embolism 1 year after operation (Table 1). Both patients were in chronic atrial fibrillation and were not anticoagulated. Actuarial freedom from emboli at 8 and 10 years is 96% ± 2% (Fig 2). Currently, 28 patients are in atrial fibrillation, 12 are in sinus rhythm, and 2 have a permanent pacemaker; 24 (57%) are receiving long-term anticoagulants.

Table 1. Major Postoperative Complications

Complication	No.	% per Patient-Year
Late death	9	2.5 ± 0.8
Thromboemboli		
Total	2	0.6 ± 0.4
Fatal	1	0.3 ± 0.3
Anticoagulant-related hemorrhage		
Total	5	1.4 ± 0.6
Major	1	0.3 ± 0.3
Fatal	. . .	. . .
Endocarditis	. . .	. . .
Paraprosthetic leak	. . .	. . .
Structural deterioration	10	2.8 ± 0.9
Overall reoperations	10	2.8 ± 0.9
Overall complications	24	6.7 ± 1.3

Anticoagulant-related hemorrhages occurred in 5 patients (1.4 ± 0.6%/pt-y); they were minor in 4 and major, requiring hospitalization, in 1 patient who sustained a gastric hemorrhage 8 months after discharge and was successfully treated. No instances of prosthetic endocarditis or paraprosthetic leak were observed.

Reoperation was required in 10 patients (2.8 ± 0.9%/pt-y) 45 to 106 months (mean, 76 ± 18 months) after the initial operation, with no deaths. The cause of reoperation was structural deterioration of the mitral LPB in all cases. Actuarial freedom from structural deterioration at 8 and 10 years is 78% ± 8% and 63% ± 11% (Fig 3).

A total of 25 complications were observed in this series (6.7 ± 1.3%/pt-y): nine late deaths, one major embolic episode, five anticoagulant-related hemorrhages, and ten reoperations because of LPB structural deterioration. Actuarial freedom from all complications at 8 and 10 years is 67% ± 8% and 53% ± 10% (Fig 4).

Pathological Findings in Explanted Liotta Bioprostheses

The most common finding was prolapse of a cusp, most frequently the right coronary one, which in some explants appeared flail because of commissural detachment (Table 2). Tears and frayings of other cusps were occasionally observed, being usually calcium-related. In one explant prolapse and perforation of the right coronary cusp was

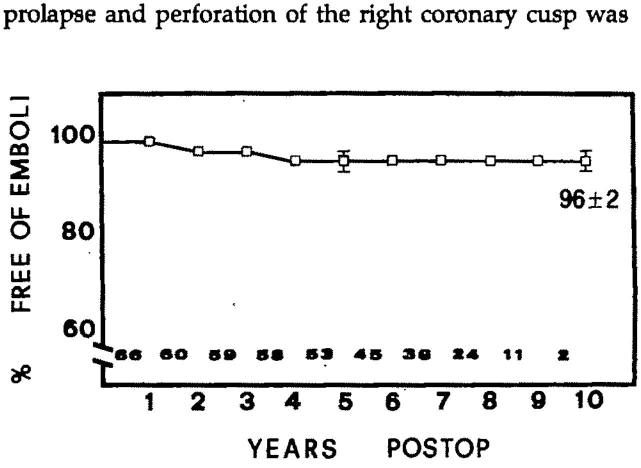

Fig 2. Actuarial freedom from thromboembolic complications.

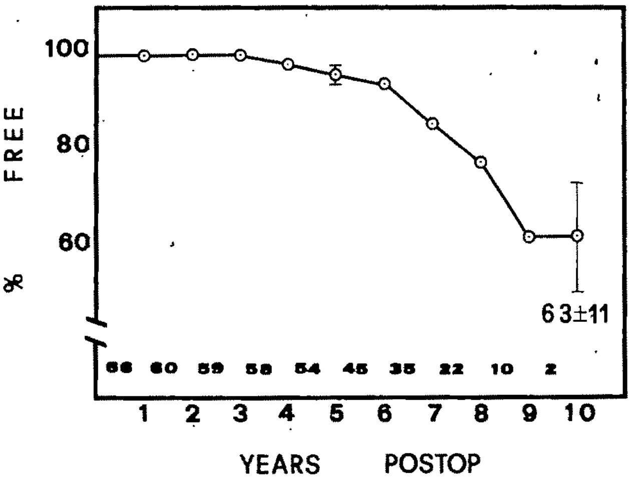

Fig 3. *Actuarial freedom from bioprosthetic structural deterioration.*

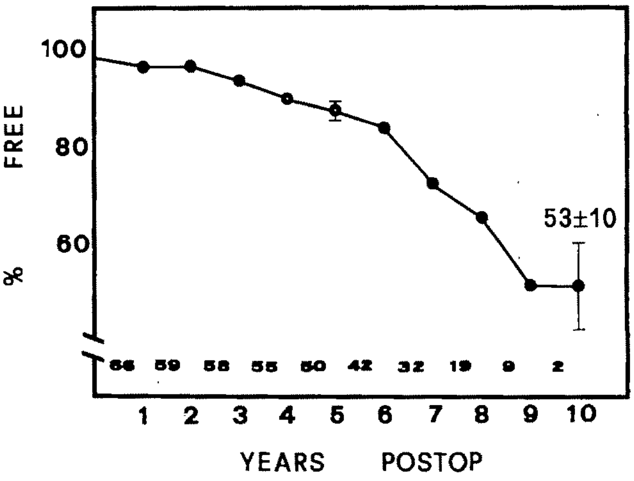

Fig 4. *Actuarial freedom from overall postoperative complications.*

present without tears. As a result of these findings the predominant mode of failure in LPB observed in this series was severe mitral regurgitation. Mild to moderate commissural and cusp calcification was detected in all explants (Figs 5, 6).

Comment

Although the LPB has been available since 1976, with approximately 10,000 implants worldwide (St. Jude Medical Technical Bulletin, 1987), reports on the clinical durability of this device are surprisingly limited. We have therefore reviewed the long-term follow-up of patients receiving an LPB at our institution to assess the overall performance of this device particularly when compared with that of standard Hancock porcine bioprostheses, more extensively used by us since the early 1970s [5].

Complications related to left ventricle–prosthesis mismatch after MVR, such as left ventricular outflow obstruction [3] and strut perforation of the left ventricular wall [4], are potential hazards with standard, high-profile Hancock valves. Therefore, the LPB has been employed in

Table 2. Clinical Data and Pathological Findings of Patients Undergoing Reoperation for Structural Deterioration of a Mitral Liotta Bioprosthesis

Patient No.	Age (y)	Sex	Months in Place	Dysfunction	Calcification[a]	Pathological Findings	Outcome
1	34	F	74	Incompetence	+	Commissural tear, prolapse of RCC, mild FTO	Alive
2	44	F	74	Stenosis, incompetence	++	Cusp rigidity, perforation and prolapse of RCC	Alive
3	52	F	92	Incompetence	+	Commissural tear, prolapse of RCC, mild FTO	Alive
4	57	F	85	Incompetence	+	Commissural tear, flail RCC	Alive
5	53	F	45	Incompetence	++	Commissural tear, prolapse of RCC	Alive
6	61	F	75	Incompetence	++	Commissural tear, flail RCC	Alive
7	47	F	64	Incompetence	+	Commissural tear	Alive
8	55	F	52	Incompetence	+	Cusp laceration, small PVL	Alive
9	38	M	106	Incompetence	++	Cusp and commissural tears	Alive
10	46	M	99	Incompetence	++	Commissural tear, flail RCC	Alive

[a] As seen by roentgenography.

FTO = fibrous tissue overgrowth; PVL = paravalvular leak; RCC = right coronary cusp; + = mild; ++ = moderate.

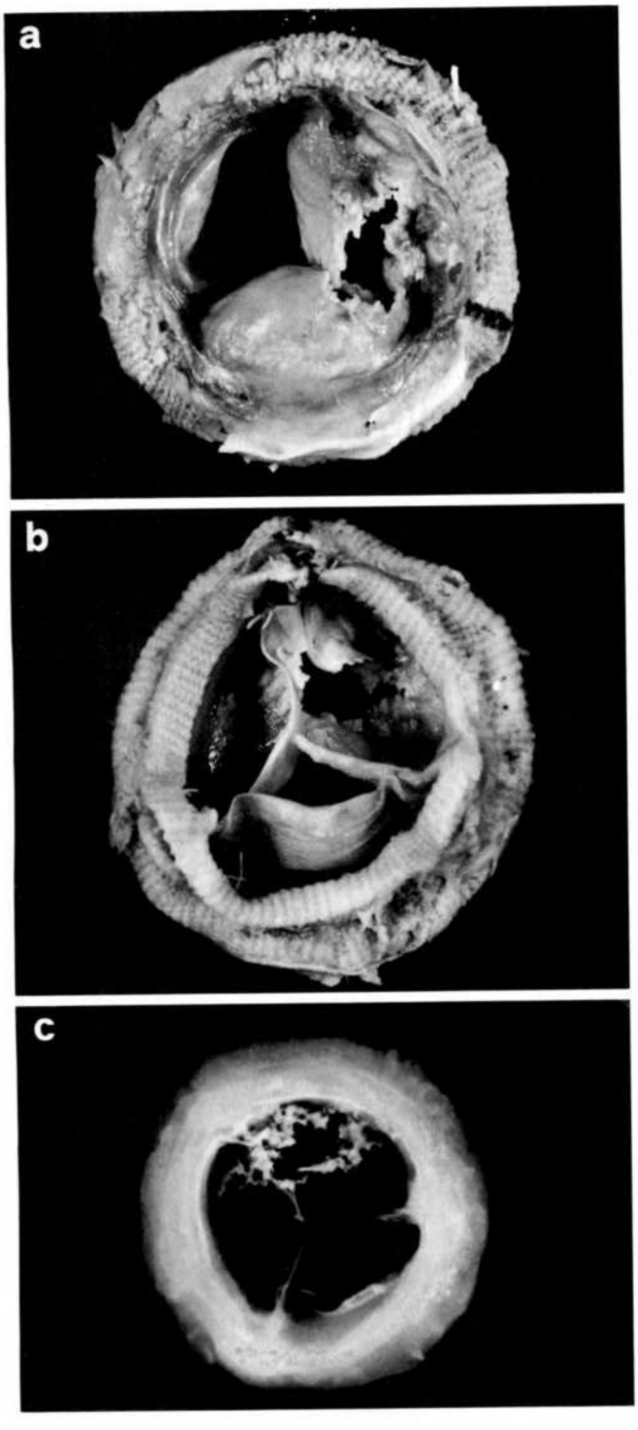

Fig 5. (Patient 9.) Gross view of a Liotta low-profile porcine bioprosthesis explanted because of severe regurgitation from a 38-year-old man 106 months after mitral valve replacement. From both the atrial (a) and ventricular (b) aspects a laceration of the right coronary cusp due to calcific deposits is evident. (c) Roentgenogram of the explant confirming the presence of calcification mainly on the right coronary cusp.

our department almost exclusively in the mitral position and in patients with a small left ventricular cavity, and such complications have been virtually eliminated. However, after our initial reports [3, 4] these hazards have not been observed even in patients receiving a Hancock porcine bioprosthesis, most likely owing to avoidance of incorrect prosthetic orientation in the mitral annulus and to proper patient selection. Furthermore, two early deaths

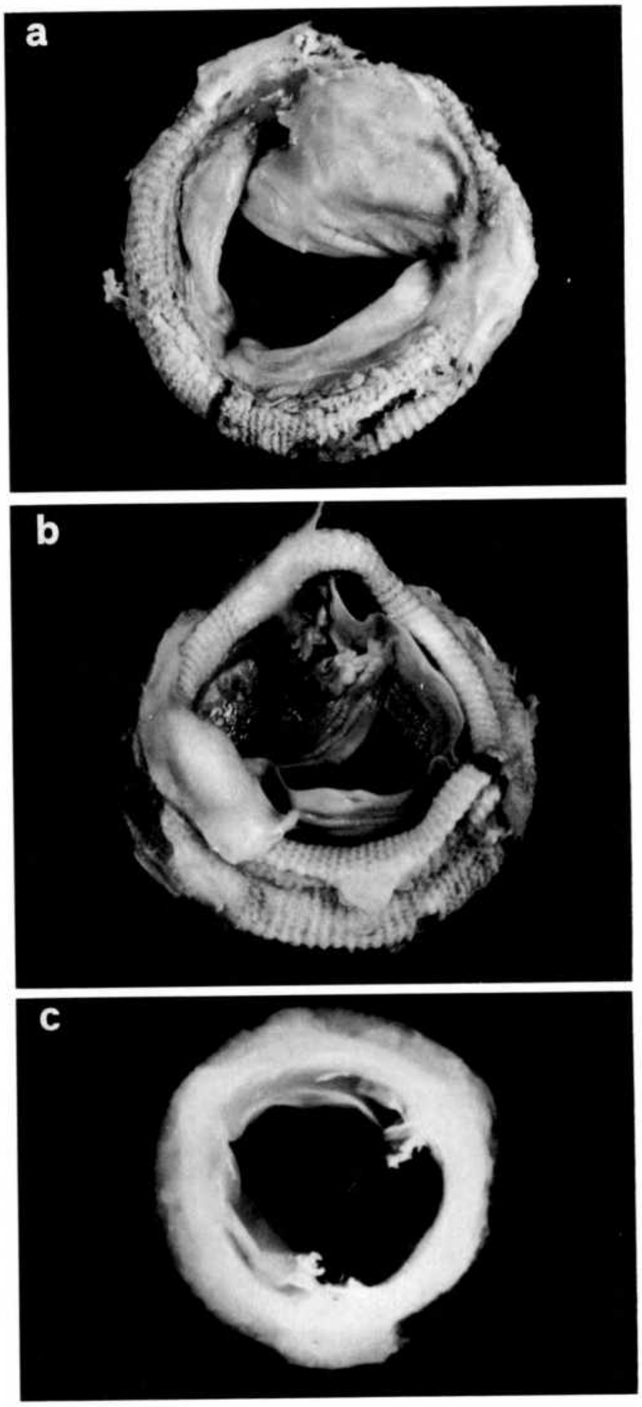

Fig 6. (Patient 10.) Mitral Liotta low-profile porcine valve explanted from a 46-year-old man after 99 months because of severe incompetence. (a) View of the atrial aspect showing prolapse of the right coronary cusp caused by rupture of the corresponding commissure due to intrinsic calcification, which is evident on the ventricular side (b). (c) Roentgenogram of the explant shows the presence of calcific deposits at the commissures.

in patients with an LPB were caused by left midventricular rupture; this type of lesion, however, is clearly not related to the type of prosthesis and appears to be prevented by preservation of the posterior mitral valve leaflet and chordae [8], not performed in those 2 patients.

Patient survival and freedom from thromboembolic events after 10 years were substantially similar to those observed by us with mitral Hancock porcine xenografts in a much larger series of patients [5]. Particularly, in our experience the LPB was associated with an extremely low incidence of thromboembolic complications after MVR; these data compare favorably with those reported by others at shorter follow-up intervals [9, 10].

Structural deterioration, mainly due to tissue calcification, is responsible for the limited durability of standard porcine bioprostheses [5, 6, 11], and this occurs also in patients with an LPB. When compared with the standard Hancock porcine bioprosthesis the LPB appears to have a higher incidence of structural deterioration. In fact, at 10 years freedom from this complication was 63% ± 11% versus 77% ± 2% for patients receiving a Hancock bioprosthesis in the mitral position as observed in our most recent survey of porcine valve recipients [11], although this difference is not statistically significant.

It has been recently demonstrated experimentally that excessive reduction of the height of the stent results in increased mechanical stresses on the closed cusps of a bioprosthesis [12]. Our pathological findings substantiate in the clinical setting the conclusions of such experimental work [13]. Owing to the unique mounting technique and frame configuration adopted to minimize the stent profile, the LPB discloses already before implantation a bulging of the cusps well above the sewing ring. As a result of the continuous closing stresses, leaflet prolapse and commissural rupture, associated with even mild degrees of calcification, occur and lead to severe prosthetic regurgitation. This mode of failure, common to all LPB explants studied, so that it could be confidently predicted in the most recent patients before reoperation, appears to be typical of this device. Whether the particular design of the LPB will result in an increasing rate of failures and in a significantly lower durability than the standard Hancock porcine valve remains to be established with progression of follow-up after the first decade.

In conclusion, although our experience is based on a limited number of patients, our results, at variance with the optimism of some medium-term reports [14], seem to indicate that the LPB does not provide any significant advantage when compared with standard porcine bioprostheses at least when used for MVR. On the other hand, the LPB appears to be even less durable, most likely as a consequence of its unique design, confirming that an excessive reduction of the stent height may favor the onset of structural deterioration and adversely affect long-term bioprosthetic performance [7, 13].

Addendum

During preparation of the manuscript another patient underwent successful reoperation to replace a failing LPB 105 months after MVR. Morphological evaluation of the valve showed structural changes similar to those of previous explants, consisting of prolapse of the right coronary cusp and rupture of one commissure associated with mild calcific deposits.

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We express our gratitude to Gian Carlo Pengo and Agostino Leorin for their skilled technical assistance.

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Surgical Management of Pulmonary Metastatic Leiomyosarcoma With Gross Endobronchial Extension

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Metastatic leiomyosarcoma occasionally is seen with gross endobronchial extension without invasion of the bronchial wall. These patients have major airway obstruction and partial or total atelectasis of the lung. Precise bronchoscopic assessment coupled with intraoperative bronchotomy allows the surgeon to determine the origin of the tumor and to save uninvolved pulmonary parenchyma. Our experience with 4 such patients illustrates the possibility of saving lung tissue despite total bronchial obstruction.

(Ann Thorac Surg 1990;50:739–42)

An important principle in the surgical management of pulmonary metastases is the conservation of any uninvolved lung. In most cases, the metastases are peripheral and resection can be accomplished with the loss of little lung tissue. In some cases, however, the tumor is more central and larger resections are required.

We have operated on 4 patients with pulmonary metastases from leiomyosarcoma who had the unique feature of massive endobronchial extension. The clinical significance of this growth pattern prompted this report.

Case Reports

Patient 1

A 53-year-old woman had a dry cough and a low-grade fever. Seven years earlier, she had undergone a total abdominal hysterectomy and a bilateral salpingo-oophorectomy for a leiomyosarcoma of the uterus.

On examination, the patient was found to have absent breath sounds over the left hemithorax. The chest roentgenogram showed opacification of the left lung with shift of the mediastinum and elevation of the left hemidiaphragm. At bronchoscopy, a firm polypoid mass was seen occluding the left main bronchus 2 cm from its origin. The tumor was assessed to be invading the anterior wall of the bronchus. Nuclear liver-spleen and bone scan results were negative. At thoracotomy, a large mass was palpated within the left lower lobe. A frozen section confirmed that the mass was a spindle cell tumor, and a left pneumonectomy was performed.

The malignancy was found to be a 9-cm metastatic leiomyosarcoma in the lower lobe with endobronchial tumor extending from the left lower lobe bronchus to the carina; notably, the bronchial wall above the site of origin within the lower lobe was not invaded. Upon reassessment, it may have been possible to save the left upper lobe if the endobronchial growth pattern had been recognized.

The patient did well postoperatively without adjuvant chemotherapy or radiotherapy and was lost to follow-up 5½ years later without evidence of metastases.

Patient 2

A 40-year-old woman had dyspnea, palpitations, and increasing chest pain. She gave a 1-month history of fever and productive cough. Six years previously, she had undergone a total abdominal hysterectomy for "fibroids." The physical examination revealed dullness to percussion and markedly decreased breath sounds over the right hemithorax. A chest roentgenogram revealed opacification of the right lung. At bronchoscopy, a polypoid mass was seen to obstruct the right main bronchus. A bronchial biopsy established the diagnosis of a malignant spindle cell neoplasm. Roentgenogram after bronchoscopy revealed reexpansion of the upper lobe and a discrete mass within the right lower lobe. Computed tomographic scans of the head and chest and a nuclear bone scan showed no metastases. At thoracotomy, multiple metastatic nodules were identified in the lower lobe. A bronchotomy was performed that confirmed the bronchoscopic impression that the endobronchial component of the tumor did not invade the wall of the bronchus intermedius. The endobronchial extension of tumor was resected en bloc with the right lower lobe. No adjuvant chemotherapy was administered. The patient died 22 months later of widespread metastatic disease.

Patient 3

A 71-year-old woman had an 8-week history of dyspnea on exertion, cough, and left-sided chest pain. Seven years previously, the patient had undergone resection of a low-grade retroperitoneal leiomyosarcoma. On examina-

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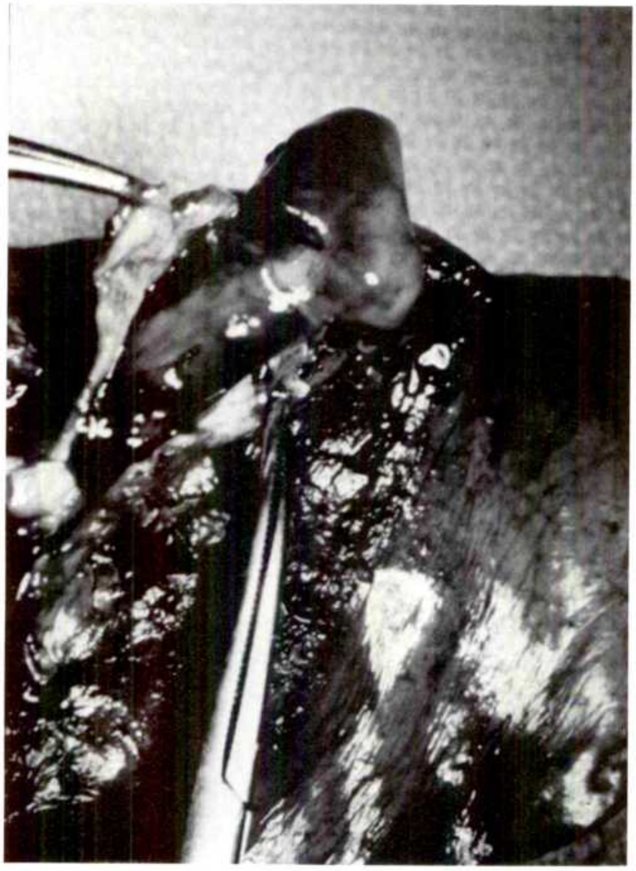

Fig 1. (Patient 3.) Operative photograph illustrating the gross endobronchial extension of a leiomyosarcoma metastatic to the left lower lobe. The endobronchial component had obstructed the left upper lobe bronchus.

tion, there were markedly decreased breath sounds over the left hemithorax. A chest roentgenogram demonstrated opacification of the left lung with mediastinal shift. A computed tomographic scan of the chest and upper abdomen failed to disclose any evidence of metastatic disease in the right chest or liver. At bronchoscopy, a glistening polypoid mass filled the left main bronchus. Large fragments of the tumor were removed, which established the diagnosis of a malignant spindle cell neoplasm and reaerated the left upper lobe. It was the bronchoscopic impression that the tumor did not invade the bronchial wall proximal to the left upper lobe orifice. At thoracotomy, a bronchotomy at the left lower lobe bronchus revealed that the 3-cm tumor originated from the superior segment with endobronchial extension but without invasion of the bronchial wall proximal to the superior segmental bronchus. A left lower lobectomy was performed (Fig 1).

Eighteen months after operation, hepatic metastases developed, which were treated with chemotherapy, and the patient died 2 years after the pulmonary resection.

Patient 4

A 42-year-old woman had a cough and mild shortness of breath. Two years prior, she had undergone a total abdominal hysterectomy and a bilateral salpingo-oophorectomy for a low-grade leiomyosarcoma with a recurrence in the ovary. Six months before the current admission, she underwent a wedge excision of a solitary and asymptomatic metastasis in the right lower lobe.

On physical examination, the patient had no appreciable air entry into the right hemithorax. A chest roentgenogram demonstrated complete opacification of the right lung with a shift of the mediastinum to that side (Fig 2A). At bronchoscopy, tumor was visualized within the lumen of the right main bronchus as a glistening, gray-white polypoid mass without recognizable attachment to the bronchial wall (Fig 2B). The right upper lobe bronchus could not be seen. At the time of thoracotomy, a 9-cm tumor was seen to largely replace the right middle lobe. A bronchotomy was performed at the level of the bronchus intermedius and demonstrated that the tumor did not invade the bronchial wall proximal to this point. A right middle and lower lobectomy was performed, and the tumor was found to arise from the spur between the right middle and lower lobes (Fig 2C). All gross disease was resected and bronchial margins were free of tumor. The postoperative chest roentgenogram demonstrated complete reexpansion of the right upper lobe (Fig 2D). The patient did well after operation with marked improvement in the dyspnea. No adjuvant chemotherapy was given. She died 7 months later from a cardiac arrhythmia without clinical evidence of tumor recurrence.

Comment

Leiomyosarcoma is an uncommon malignancy usually arising in the uterus or in the retroperitoneum. The differential diagnosis between a leiomyosarcoma and simple degeneration of a uterine leiomyoma may be difficult, and in 2 of our patients, the primary tumors were originally misdiagnosed. These sarcomas tend to metastasize hematogenously to the lungs and liver. Because the diagnosis of the primary tumor is occasionally erroneous and because there is often a prolonged disease-free interval, the clinician may not be alert to this diagnostic possibility.

The pulmonary metastases are usually large and limited in number. The tendency of these tumors to extend into the pulmonary vein has been recognized and should be considered when performing a resection [1]. The gross extension through the bronchus to obstruct a main bronchus without direct involvement, however, has not been adequately appreciated. In our experience, leiomyosarcomas also may extend from the pulmonary parenchyma through the airway for a considerable distance without involving the bronchial wall. This growth pattern has been described in both metastatic and primary leiomyosarcomas of the lung [2–4]. The bronchoscopic appearance in the former is that of an endobronchial polypoid mass without involvement of the bronchial wall. It is important to assess the origin of the tumor mass as far distal as possible because this determines the site for an intraoperative bronchotomy. Such a bronchotomy was per-

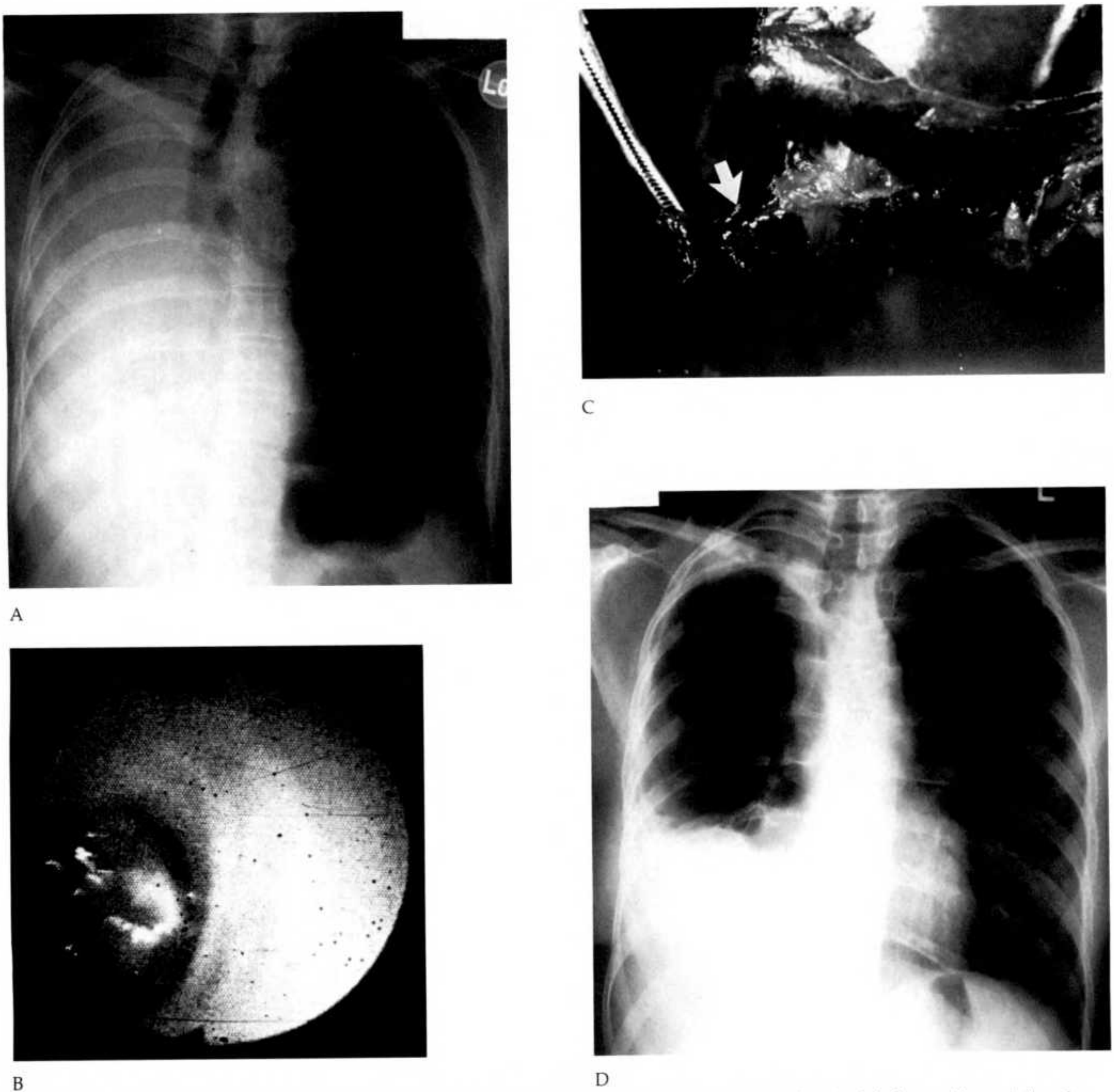

Fig 2. (Patient 4.) (A) Preoperative posteroanterior chest roentgenogram demonstrating a completely atelectatic right lung with no air bronchogram. (B) Endoscopic appearance of the tumor in (A) with endobronchial extension of tumor up to the level of the right main bronchus but without invasion of the bronchial wall. (C) Operative photograph demonstrating endobronchial tumor extending above the bronchus intermedius (arrow). *(D) Postoperative posteroanterior chest roentgenogram with reexpansion of the right upper lobe.*

formed to determine the exact origin of the tumor in the 3 patients in whom lung parenchyma was saved.

A distinction must be made between these tumors and true endobronchial metastases, which arise in the bronchial wall. The latter tumors are usually limited in size at the time of clinical presentation and lend themselves to endobronchial laser or brachytherapy salvaging most of the obstructed lung [5, 6]. Carcinomas of the breast, kidney, and colon and melanomas are the most likely primary sites for endobronchial metastases [7–10]. However, in patients with metastatic leiomyosarcoma with gross endobronchial extension, the endobronchial component is bulky and part of the distal atelectatic lung usually is replaced with tumor, making laser photoablation and brachytherapy unfavorable modes of therapy.

Because most of the bronchial wall is uninvolved, surgical resection with conservation of the uninvolved lung parenchyma is the procedure of choice. This distinction between endobronchial extension and true metastases to the bronchial submucosa must be made on bronchoscopy, which should be performed before resection of metastatic sarcoma with bulky tumor load, and radiologic or clinical evidence of airway obstruction.

Previous reports of metastatic leiomyosarcoma with endobronchial extension emphasize the potential for the development of obstructive pneumonia, airway bleeding, and cardiac arrhythmias secondary to hypoxia in these patients [2, 3]. As these sarcomas tend to be unresponsive to either chemotherapy or radiotherapy, we agree with others that resection of the metastases, while conserving obstructed but otherwise uninvolved lung tissue, should be performed for the palliation of these airway symptoms [11–14]. Adjuvant chemotherapy after complete surgical resections has not been found to be of benefit. In none of our 4 patients did local recurrence develop, and 1 long-term survivor is reported.

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Clinical Performance of St. Jude and Medtronic-Hall Prostheses: A Randomized Comparative Study

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Newer and improved models of mechanical prostheses are regularly added to surgeons' armamentarium. This study was designed to compare the clinical performance of two of the most used current models of mechanical prostheses. From August 1983 through July 1985, 182 white patients were prospectively randomized to implantation of the St. Jude Medical (95 patients) or Medtronic-Hall (87 patients) prostheses. There were 84 mitral, 85 aortic, and 13 double (mitral and aortic) valve replacements. There were no differences between the two groups with regard to sex distribution, age, functional class, emergency operation, and site of implantation. Early mortality was 3.2% for patients with the St. Jude valve and 5.7% for those with the Medtronic-Hall (p = NS). The survivors were followed for 3 to 5 years (mean, 4.2 ± 0.6 years; cumulative follow-up, 559 patient-years). Late mortality was 7.1%/patient-year for the St. Jude group and 3.2%/patient-year for the Medtronic-Hall group ($p < 0.05$). However, the valve-related mortality was equal (1.4%/patient-year) for both groups. Noncardiac causes accounted for most of the difference between the St. Jude and Medtronic-Hall groups (2.5%/patient-year and 0.4%/patient-year, respectively). There were no cases of thrombotic obstruction, whereas serious systemic thromboembolism occurred at the rate of 1.8%/patient-year (5 episodes) for the St. Jude group and 2.5%/patient-year (7 episodes) for the Medtronic-Hall group (p = not significant); there were another nine episodes of systemic embolism that left no sequelae. Three patients (St. Jude, 2; Medtronic-Hall, 1), all of whom had aortic valve replacement, required reoperation (0.5%/patient-year) because of prosthetic endocarditis, with two deaths. Actuarially, 74% of St. Jude and 80% of Medtronic-Hall patients survived 5 years and 92% and 89%, respectively, were free from systemic thromboembolism (p = not significant). At a medium-term follow-up, these two mechanical valves appear to have similar performances, which compare favorably with those of other devices used previously.

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There is still a good deal of controversy and confusion about the qualities, or otherwise, of the cardiac valve prostheses in current clinical use. The multitude of models available for implantation is the clearest indication of the limitations of each and every one of them; the ideal prosthesis remains an elusive goal.

After a decade of widespread acceptance, bioprostheses are gradually losing the favor of many surgeons throughout the world. Our own experience confirmed a limited durability, especially in very young patients in whom early calcification led to disastrous results [1], and the use of these prostheses is now preferentially reserved for older patients and those with a life expectancy of less than 10 to 12 years. From 1980 we had been using them almost exclusively in the elderly patient in whom the risks of anticoagulation, obligatory with mechanical prostheses, were thought to be higher. However, we have recently demonstrated excellent performance of mechanical prostheses in this group of patients, with a lower incidence and morbidity of thromboembolic phenomena and only a slightly increased incidence of serious hemorrhagic episodes [2].

Among the newer generation of mechanical prostheses are the St. Jude Medical bileaflet and the Medtronic-Hall tilting-disc valves. In Johannesburg, we have now accumulated the experience of the implantation of nearly 2,000 of each, initially in two different population groups of widely divergent characteristics [3, 4]. Naturally, comparison of the respective performances was impossible; hence a randomized study was planned for each one of the two groups of patients, of which the preliminary results, largely inconclusive, were published earlier [5]. In this paper we report on the 3- to 5-year results of this study in one, the most sophisticated and compliant, of these groups.

Material and Methods

Patient Population

From August 1983 through July 1985, 182 white patients were consecutively and prospectively randomized to implantation of either the St. Jude Medical or the Medtronic-Hall prostheses in Johannesburg, South Africa. Only patients who had previous valve operation (19 patients), those whose valves were considered adequate for valvoplastic procedures (7 patients), and those who had absolute contraindications to anticoagulation (3 patients) were excluded from randomization, which was done by blind drawing of a card from a mixture of an identical number of cards for each of the two valves.

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Table 1. Summary of Clinical Data

Variable	St. Jude Medical	Medtronic-Hall
No. of patients		
MVR	44	40
AVR	45	40
DVR	6	7
Total	95	87
No. of valves	101	94
Sex (M:F)	44:51	45:42
Age (mean ± SD; y)	52.7 ± 13.6	54.0 ± 14.9
Functional class (mean ± SD)	3.1 ± 0.4	3.1 ± 0.4
Infective endocarditis (%)	5.3	6.9
Emergency (%)[a]	6.3	12.6

[a] $p < 0.05$; others, p = not significant.

AVR = aortic valve replacement; DVR = double-valve replacement; MVR = mitral valve replacement; SD = standard deviation.

The clinical characteristics of the population are summarized in Table 1. Ninety-five patients received 101 St. Jude valves and 87 received 94 Medtronic-Hall valves. The distribution of each of the two types of prostheses as to the site of implantation was similar. There were no cases of tricuspid valve replacement and 10 patients (5.5%) had tricuspid annuloplasty. The mean age (± standard deviation) of the whole group was 53.2 ± 14.7 years and was also identical for the St. Jude and for the Medtronic-Hall patients. There was an even distribution of sexes in the whole population and in the two groups. Likewise, there were no differences in the preoperative functional classification of the patients or in the prevalence of infective endocarditis or other types of pathology. However, a greater number of patients of the Medtronic-Hall group had emergency operations (11 patients; 12.6%) than in the St. Jude group (6 patients; 6.3%) ($p < 0.05$). Stenotic lesions predominated (67.2%) in patients subjected to aortic valve replacement (mainly senile calcific degeneration), whereas in those subjected to mitral valve replacement there was an even distribution of stenotic and regurgitant lesions, but there were no differences between the two groups.

All procedures were performed by the same group of surgeons, using similar techniques. Aortic valve replacements were done with interrupted figure-of-8 sutures of 2-0 Ti-cron (Davis & Geck, Danbury, CT) and mitral valve replacements with continuous sutures of the same material, except in cases of extreme calcification of the annulus, where interrupted sutures were also used. Prophylatic anticoagulation with warfarin sodium was carried out in all patients and supervised by the medical staff. The control of therapy was made by monthly analysis of the prothrombin index, which was kept at 35% to 45%. The compliance was generally good and the therapeutic ranges were obtained in most instances.

The follow-up data were collected by a social worker with nursing qualifications by thorough revision of the outpatient files and/or through a written questionnaire to the patients or their cardiologists (to the end of August 1988). The patients had been followed for a cumulative 559 patient-years with a mean of 4.2 ± 2.1 years per patient (St. Jude, 4.3 ± 2.1; Medtronic-Hall, 4.2 ± 1.9; p = not significant [NS]). Thirteen patients (7.1%) were lost to follow-up or had emigrated from the country, 6 (6.3%) in the St. Jude group and 7 (8.0%) in the Medtronic-Hall group (p = NS).

Statistical Analysis

The definitions of the events, their classification, and the analysis of the data followed the recently suggested "Guidelines for reporting morbidity and mortality after cardiac valvular operations" [6]. All events were considered in the calculation of the linearized incidences, expressed in percentage per patient-year of follow-up. Comparison of the results of the two groups was made by the χ^2 method and with the use of 2×2 contingency tables and the Yate's correction for continuity for small numbers. Values of p less than 0.05 were considered to indicate statistical significance. Actuarial curves were constructed using the methods described by Grunkemeier and Starr [7], but no patient was considered at risk for more than a single event of each type.

I am aware of the fact that valve location influences the incidence of valve-related complications after operation. However, the number of patients in each set, especially double-valve replacement, is relatively limited and could preclude attaining a reasonable level of statistical power to reject or accept conclusively the null hypothesis. As there were no differences between the two valves, as far as the number of patients is concerned, the analysis of each group as a whole and comparison between the two should prove meaningful.

Results

Mortality and Survival

Eight patients (4.4%) died early, 3 in the St. Jude group (3.2%) and 5 in the Medtronic-Hall group (5.7%; p = NS). None of the early deaths was due to valve-related causes. Twenty-nine patients (5.2%/patient-year) died late, 20 in the St. Jude group (7.1%/patient-year) and 9 in the

Table 2. Causes of Late Mortality

	St. Jude Medical		Medtronic-Hall		
Cause	No.	%/pt-y	No.	%/pt-y	p Value
Cardiac					
Valve-related	4	1.4	4	1.4	NS
Nonvalve-related	8	2.8	3	1.1	<0.05
Noncardiac	7	2.5	1	0.4	<0.02
Unknown/sudden	1	0.4	1	0.4	NS
Total	20	7.1	9	3.3	<0.05

pt-y = patient-year; NS = not significant.

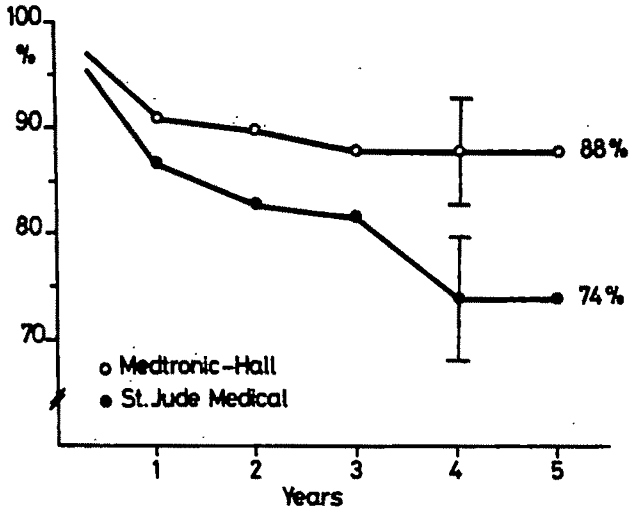

Fig 1. Actuarial global survival of patients subjected to valve replacement with Medtronic-Hall and St. Jude Medical prostheses (p < 0.05).

Medtronic-Hall group (3.3%/patient-year; $p < 0.05$). However, valve-related mortality occurred at the same rate of 1.4%/patient-year in both groups (Table 2). The difference in the global mortality rates is justified by much higher incidences of cardiac, but nonvalve-related, and noncardiac deaths in the St. Jude group ($p < 0.02$). The causes of valve-related mortality were three episodes of systemic thromboembolism and one of infective endocarditis with periprosthetic leakage in each group. The actuarial survival after 5 years was 74% ± 6% for St. Jude patients and 88% ± 5% for Medtronic-Hall patients ($p < 0.05$) (Fig 1). However, freedom from valve-related mortality was 93% ± 3% and 94% ± 3%, respectively (p = NS) (Fig 2).

Thromboembolism

Major systemic thromboembolism occurred at a rate of 1.8%/patient-year in the St. Jude group (5 cases) and of 2.5%/patient-year in the Medtronic-Hall group (7 cases; p = NS). As stated in the previous section, there were three fatal thromboembolic events in each group; the remaining 2 patients with St. Jude valves (0.7%/patient-year) and 4 with Medtronic-Hall valves (1.5%/patient-year) had lasting sequelae. Besides the 12 patients who had serious thromboembolic episodes, another 9 (7 with St. Jude [2.5%/patient-year] and 2 with Medtronic-Hall [0.7%/patient-year] valves) had transient neurological events that could have been of thromboembolic origin (Table 3). There were no cases of known thrombotic obstruction in this study, but 1 patient in each group died suddenly and unexpectedly of an unknown cause. Freedom from major systemic thromboembolism was 92% ± 4% and 89% ± 4% for St. Jude and Medtronic-Hall patients, respectively, after 5 years (p = NS) (Fig 3).

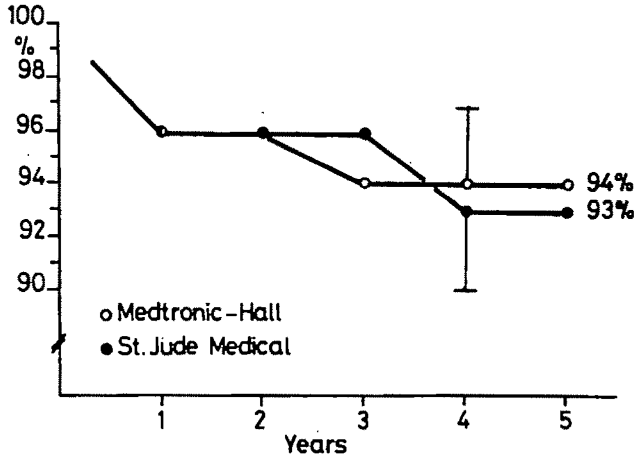

Fig 2. Actuarial estimate of freedom from valve-related mortality (p = not significant).

Table 3. Incidence of Valve Thrombosis and Systemic Thromboembolism

	St. Jude Medical		Medtronic-Hall		
Event	No.	%/pt-y	No.	%/pt-y	p Value
Valve thrombosis	0	. . .	0	. . .	
Thromboembolism					
Fatal	3	1.1	3	1.1	NS
With sequelae	2	0.7	4	1.5	NS
No sequelae	7	2.5	2	0.7	<0.05
Total	12	4.3	9	3.3	NS

pt-y = patient-year; NS = not significant.

Other Complications

Three patients, 2 in the St. Jude group (0.7%/patient-year) and 1 in the Medtronic-Hall group (0.4%/patient-year; p = NS), were reoperated on as emergencies and in New York Heart Association class IV because of prosthetic valve endocarditis. Two of these patients (66%), 1 in each group, died. The second patient in the St. Jude group survived the reoperation and was cured of his infection.

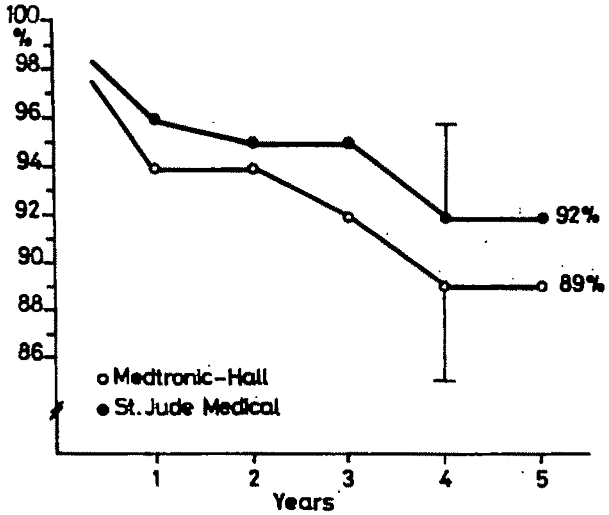

Fig 3. Probability of survival free from major systemic thromboembolism (p = not significant).

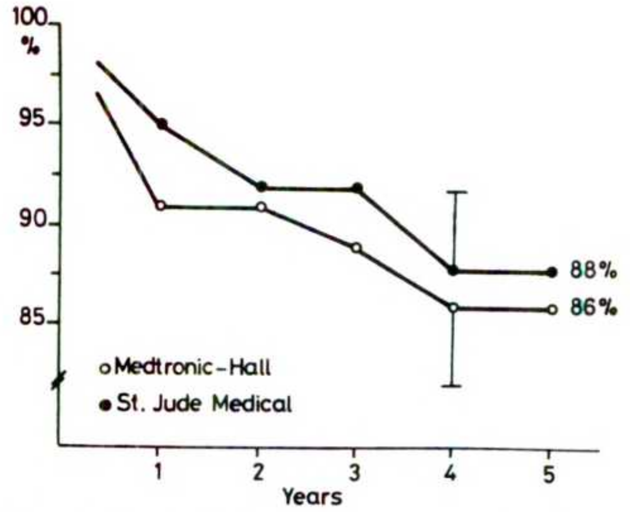

Fig 4. Actuarial survival free from all valve-related complications (mortality and morbidity) (p = not significant).

There were no serious hemorrhagic events, but 2 patients of each group had serious bleeds that did not require any type of treatment. There were no cases of structural or of nonstructural prosthetic dysfunction. In actuarial terms, 88% ± 4% of the patients with St. Jude valves and 86% ± 4% of those with Medtronic-Hall valves were free from all valve-related complications after 5 years (Fig 4).

Comment

The frequent alterations introduced in the mechanical characteristics of the different prostheses may make comparative studies superfluous in the long-term, as the models originally used may no longer exist. Hence the paucity of such studies, whether randomized or not [8–11], despite the fact that more than 1,000,000 cardiac valve prostheses of many models have been used throughout the world after the first implantation by Harken and colleagues in 1960. Fortunately, however, both the Medtronic-Hall and the St. Jude valves have suffered virtually no changes since their appearance in 1977 and 1978 and remain unmodified since this study was commenced in 1983.

In Johannesburg we have had the experience of implantation of nearly 2,000 of each of these two prostheses. However, the fact that they were initially used in two population groups with widely different characteristics was an obstacle to making any conclusions about their comparative clinical performances. Therefore, a prospective randomized comparative study of these two prostheses and in both population groups was conceived and carried out from 1983 through 1985. The first, short-term analysis of the results of this study [5] was largely inconclusive with disparate indexes of performance of the two valves. As the different complications of prostheses do not occur at a constant rate, analysis of the results after a short period of follow-up may have led to cross-sectional errors because of the inclusion of complications of earlier occurrence and of the exclusion of those occurring at a later stage.

It was never our aim to evaluate the hemodynamic properties of the valves, as these have been exhaustively studied by others. Rather, this study was directed at comparing their clinical performances. The clinical data confirm the adequacy of randomization. The differences in the late mortality rates of the St. Jude group cannot be attributed to valve-related causes, the incidence of which was similar in both groups. The higher incidence of other cardiac and of noncardiac deaths, mostly of neoplastic and traumatic origin, is unexplained, but may have been due to chance alone.

Thromboembolic complications are still the most important causes of morbidity and even of mortality with mechanical valves and are most relevant in the analysis of the performance of any mechanical prosthesis. Based on the incidences previously described from this Unit in a similar group of patients [3], we would have expected 3 or 4 cases of thrombotic obstruction. Surprisingly, none has occurred, although this complication could have been the cause of the two sudden, unexplained deaths. In contrast with the opinion expressed by other authors [12], in our past experience fatal thrombosis has often occurred with few prodromal symptoms or physical signs. Also, our experience appears to indicate a higher incidence of valve thrombosis within the first 5 years after implantation (Antunes MJ, unpublished data). Thereafter it decreases, probably as the sewing ring becomes endothelialized.

Recently, Butchart and associates [12] have reported on a large series of patients with Medtronic-Hall prostheses followed up for a total of 2,640 patient-years, also with a zero incidence of prosthetic thrombosis, which the authors attributed not only to the favorable characteristics of this valve but also to the use of better controlled, low-intensity anticoagulation. By contrast, their experience with the Lillehei-Kaster and the Omniscience valves revealed strikingly higher incidences of thrombotic obstruction. We had also shown a lower rate of thrombosis with the Medtronic-Hall valve than with other valves in our black population group [4]. On the other hand, many series published in the past 5 years demonstrated low incidences of both thrombosis and systemic embolism with the St. Jude Medical valve [13–15]. Our own experience with this device, in the same population group in which the current study was performed, testifies to these low rates, especially when compared with previous experiences with other valves [3].

In the current series, the incidence of serious systemic thromboembolism was only slightly and not significantly higher in patients with the Medtronic-Hall valve. Therefore, the difference observed in the earlier follow-up study [5] seems to be diluting because of a decreased incidence in late follow-up. In other words, systemic thromboembolism occurred earlier with the Medtronic-Hall valve and later in the St. Jude group, which may be explained by the influence that the differences in the engineering characteristics of the two valves have in the genesis of thrombotic material and its subsequent embolization. Notably, the incidence of fatal thromboembolism was now similar with both prostheses.

Conversely, there is anticoagulant-related hemorrhage, the inverse relationship of which with thrombosis and thromboembolism led to the suggestion that the three complications should be lumped together as one of the indexes of valve performance [16]. In the current series there were no fatal or even serious hemorrhagic episodes. By contrast, Butchart and associates [12] found an incidence of 0.2%/year of fatal bleeding with what they considered low-intensity anticoagulation. Obviously, it is extremely difficult to compare the intensity of anticoagulation in different series of patients. Besides the prosthetic design and manufacturing characteristics, other factors may influence the incidence of valve thrombosis and systemic thromboembolism, including the adequacy of anticoagulation and respective patient compliance, the pathology involved, and the surgical and suture techniques [17–19]. Naturally these factors vary from series to series and comparisons of results are, therefore, inappropriate. This study has succeeded in eliminating biases by these factors; any differences encountered are, therefore, likely to be related to the characteristics of the prostheses alone.

In summary, the results obtained from this study appear to indicate that, in the medium term, the performances of the St. Jude and Medtronic-Hall prostheses are identical in this compliant population group and compare very well with those reported recently by other investigators and by ourselves. I believe that these two valves are among the best of the modern generation of prostheses and, therefore, recommend them as valve substitutes when required. The choice of one or the other is, then, a matter of preference or may be linked to other factors such as easy and regular availability and price.

The St. Jude valve, with its truly low profile even in the open position, may have a slight advantage for use in the very narrow aortic root or in the small left ventricle. On the other hand, the Medtronic-Hall valve is, in my view, easier to implant and to assess clinically and echocardiographically [20], which may translate into substantial advantages to the patient.

I acknowledge the essential role of Drs R. Kinsley and P. Colsen and the other surgeons of the Division of Cardiothoracic Surgery of the Johannesburg Hospital and the University of the Witwatersrand, Johannesburg, South Africa, in the conception and performance of this study. I also express my gratitude to Prof S. Cronje, current Head of the Division, for the permission to publish this material, and to Anne Betts for collecting the follow-up data and to Ana Maria Alves for typing the manuscript.

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Clinical Experience With the Medtronic-Hall Valve Prosthesis

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Medtronic-Hall valves were implanted during 204 procedures performed between 1982 and 1988. Mean population age was 54.4 years; 96% of patients were in New York Heart Association functional class III or greater. Emergency operations constituted 16% of the procedures. Rheumatic heart disease was the single most common indication for valve replacement. In 18% of patients, operation was performed to replace a previous prosthetic valve. The mean follow-up was 3.2 years. Overall operative mortality was 10.3%, the highest mortality being for double-valve replacements (24%). Valve-related mortality, by position, was 5.3% for aortic valves, 6.0% for mitral valves, and 4.0% for multiple-valve replacements. Actuarial 5-year freedom from events were: survival, 68%; thromboembolism, 90%; prosthetic valve endocarditis, 98%; paravalvular leak, 95%; and reoperation, 92%. Complications with the highest mortality were thromboembolism (36%) and endocarditis (33%). The complication rates in this series are high but the patients were more severely ill than in other reports, and operative survivors experienced a considerable improvement in New York Heart Association functional class.

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Mechanical valve prostheses consistently demonstrate superior durability yet they remain plagued by the persistence of thromboembolic complications that have been the focus of alterations in the design of newer models. When the Medtronic-Hall tilting-disc valve was introduced the improvements in design over its tilting-disc predecessors included an increased opening angle, two-phase movement of the disc on a central strut, and a larger minor orifice [1]. These changes were made to improve the flow characteristics and turbulence, thereby reducing the incidence of thromboembolism.

Published experience with the Medtronic-Hall valve is not extensive [2–5]. To date there have been few studies originating in North America [6]. Since 1982 the Medtronic-Hall valve has been the mechanical prosthesis of choice at the University of Pittsburgh. This report documents our intermediate experience with this valve.

Material and Methods

Patients

From April 1982 through March 1988, 231 Medtronic-Hall (MH) valve prostheses were implanted in 197 patients during 204 procedures. Isolated mitral valve replacement (MVR) was performed in 84 patients, isolated aortic valve replacement (AVR) was performed in 95 patients, and 24 patients underwent double-valve replacement (DVR) with the MH valve prosthesis (19 aortic and mitral; 3 aortic, mitral, and tricuspid; and 2 mitral and tricuspid). There was also 1 case of isolated tricuspid valve replacement. Overall, 117 MH valve prostheses were implanted in the aortic position, 108 in the mitral position, and 6 in the tricuspid position during 204 operations. Patient age ranged from 13 to 81 years with a mean of 54.4 ± 15.4 years. Seventy percent of the patients were 50 to 79 years old. There was a female predominance in the MVR and the DVR groups and there were more male patients in the AVR group (Table 1). Twenty-nine patients (35%) in the MVR group and 10 (42%) in the multiple replacement group were in atrial fibrillation preoperatively. One hundred nine patients (53%) were in New York Heart Association (NYHA) functional class IV, 88 (43%) were in class III, and 7 (3%) in class II. Previous valve procedures had been performed on 13% of mitral valve patients, 21% of aortic valve patients, and 28% in the multiple valve group. Elective operations were performed on 171 (84%) patients whereas 33 (16%) were done as urgent or emergency procedures. Rheumatic disease, nonrheumatic calcific valvular disease, and replacement of dysfunctioning prosthetic valves were the most common preoperative pathologies and accounted for 64% of the indications for operation (Table 2). Coronary artery bypass grafting was concomitantly performed in 2/24 multiple valve replacements, in 17/95 AVRs, and in 13/84 MVRs. Other associated procedures were performed in 14% of patients. Tricuspid valve replacement or repair was required in 9/84 MVRs and in 5/24 multiple valve replacements.

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Table 1. Operative Procedures and Patient Demographics

Variable	MVR	AVR	DVR	TVR	Total
Patients (No.)	84	95	24	1	204
Valves (No.)	84	95	51[a]	1	231
Sex (M:F)	1:2	4:1	1:1.5		1:2
Mean age (y)	55.4	53.6	54.0		54.4
Atrial fibrillation (%)	34.5	10.5	41.6		24
NYHA class (%)					
II	1	6			3
III	43	46	32		43
IV	56	48	68		53
Emergency (%)	16	11	36		16
Previous valve operation (%)	13	21	28		18
Associated operation (%)	17	8	25		14

[a] Includes 24 mitral, 22 aortic, and 5 tricuspid valves.

AVR = aortic valve; DVR = multiple valve; MVR = mitral valve; NYHA = New York Heart Association; TVR = tricuspid valve.

Operative Techniques

All operations were performed with standard cardiopulmonary bypass with moderate hypothermia (25° to 28°C). Arterial cannulation was performed through the ascending aorta and venous cannulation through the right atrium. Cold blood cardioplegic solution for myocardial protection was administered through the aortic root. In the presence of aortic insufficiency or when additional doses of cardioplegia were needed and the aortic root was opened, the coronary orifices were perfused directly. Additional protection was obtained by topical cooling of the pericardial cavity with normal saline solution at 4°C. Valves were secured with simple vertical or pledgeted mattress sutures. The major valve orifice was positioned posterolaterally toward the noncoronary sinus for aortic valves; mitral valves were oriented to avoid impingement by papillary muscles or chordae tendineae. The size distribution of valves implanted is shown in Figure 1. All patients received cefamandole perioperatively.

Postoperative Management and Follow-up

Anticoagulant therapy with warfarin was begun after the chest drains were removed. Dosage was adjusted to maintain a therapeutic prothrombin time 1.5 to 2.0 times control values. Antiplatelet agents were also given to patients undergoing concomitant coronary artery bypass grafting.

Table 2. Indications for Valve Replacement

Indication	MVR	AVR	DVR	Total
Rheumatic valve disease (%)	37	11	47	28
Calcific valve disease (%)	11	31	4	18
Replace previous valve (%)	13	21	28	18
Other (%)	39	37	21	36

AVR = aortic valve; DVR = multiple valve; MVR = mitral valve.

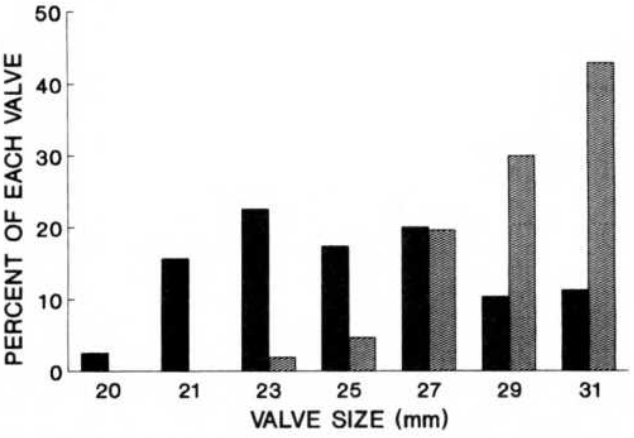

Fig 1. Distribution of valve sizes implanted in the aortic (shaded) *and mitral* (hatched) *positions. The* percent of each valve *legend refers to the percent of valves implanted in each position that were of the specified size.*

Patients surviving the operative period were followed up for a minimum of 3 months and a maximum of 5.92 years (mean, 3.2 years). The cumulative follow-up was 566 patient-years (MVR, 207; AVR, 296; DVR, 63 patient-years). All patients were followed up as outpatients in the early postoperative period with later follow-up by telephone. Ten patients (5%) were lost to follow-up during the study interval.

Definitions

The following definitions were used in accordance with previously published guidelines [7].

EARLY AND LATE MORTALITY. Operative mortality was defined as death occurring during the hospitalization in which the valve replacement was performed or deaths occurring within 30 days of the operation for those patients discharged from the hospital within the first 30 postoperative days (unless the cause of death was clearly unrelated to the operation). Late mortality included all deaths not considered operative mortality. Data regarding survival and valve-related events were obtained from review of the clinical files.

VALVE-RELATED MORTALITY. Valve-related mortality was defined as death caused by structural deterioration, nonstructural dysfunction, thromboembolism, anticoagulant-related bleeding or prosthetic endocarditis, or death at reoperation.

THROMBOEMBOLISM. Thromboembolism was defined as any new focal, neurological, or peripheral deficit, either transient or permanent, unless another source of embolism could be proved.

THROMBOTIC OBSTRUCTION. Thrombotic obstruction included all confirmed cases diagnosed at reoperation or autopsy and all clinically suspected cases in the absence of autopsy confirmation.

PROSTHETIC VALVE ENDOCARDITIS. Prosthetic valve endocarditis included any infection involving a heart valve substitute. The diagnosis was based on appropriate clinical criteria and/or histological confirmation at reoperation or autopsy.

PARAVALVULAR LEAKAGE. Paravalvular leakage was defined as valvular regurgitation whose cause was not intrinsic to the valve itself. All cases diagnosed clinically (echocardiogram, angiography) and that required reoperation because of hemodynamic imbalance are included.

NONSTRUCTURAL DYSFUNCTION. Nonstructural dysfunction was defined as valve dysfunction caused by entrapment of suture or other material and diagnosed at reoperation or autopsy.

REOPERATION. Reoperations included those performed for repair or replacement of a previously implanted MH prosthesis.

Statistical Analysis

All episodes were included in the calculation of the linearized incidence, expressed as the number of events per 100 patient-years. The actuarial probabilities of survival were calculated according to published methods [8]. Statistical significance was established by the Lee-Desu statistic [9] at a p value of 0.05.

Results

Operative Mortality

Overall operative mortality was 10.3% (21/204). The operative mortality rate by valve position was 4.2% for AVR, 13.1% for MVR, and 24% for multiple-valve replacement. Overall mortality rate for elective valve replacement was 5.3% (9/171) whereas that for emergency procedures was 36.4% (12/33). Patients undergoing isolated valve replacement had significantly lower mortality (3.4%, $p < 0.05$) with rates of 0% for AVR alone, 5.3% for MVR, and 11.1% for DVR when compared with patients having combined procedures.

The most common cause of operative death was low output syndrome. Ten patients died as a result of perioperative low output syndrome (5 multiple, 2 AVR, 3 MVR). Fatal dysrhythmias developed in 3 patients undergoing MVR. Two patients undergoing MVR died as a result of intraoperative left ventricular perforation. One patient died of a myocardial infarction after MVR and coronary artery bypass grafting. Sudden cardiac arrest in 1 patient after AVR was secondary to valve occlusion by loose suture material. One patient who underwent AVR died of prosthetic valve endocarditis and 1 patient who underwent MVR died as a result of a cerebral embolus. One patient undergoing multiple valve replacement died of cardiac tamponade and 1 undergoing MVR died of multisystem organ failure after several hypotensive episodes.

Late Mortality

Late deaths occurred in 28 patients (13.7%). The mortality rates for AVR, MVR, and DVR were 13.7%, 15.5%, and 8% respectively (Table 3). Valve-related complications were the cause of death in 11 patients for a mortality rate of 5.4%. Individual valve-related late mortality was 5.3% for AVR, 6.0% for MVR, and 4.0% for DVR patients. Other cardiac causes resulted in 9 deaths. Sudden death occurred in 2 patients and was assumed to be valve-related. Two patients died of unknown causes.

Table 3. Causes of Late Mortality ($n = 28$)

Cause	MVR	AVR	DVR	Total
Thromboembolism	2	3		5
Valve thrombosis		1		1
Cancer	1	4		5
Congestive heart failure	3	1		4
Myocardial infarct	1	1	1	3
Paravalvular leak		1	1	2
Dysrhythmia	1	1		2
Sudden death	2			2
Multisystem organ failure	2			2
Unknown	1	1		2

AVR = aortic valve; DVR = multiple valve; MVR = mitral valve.

Valve-Related Mortality

Fifteen patients (7.4%) died of complications directly related to the MH valve. The most common cause of death (n = 5) was from complications secondary to cerebral vascular accidents. Two deaths each were due to left ventricular perforation and paravalvular leakage. One death each was due to valve thrombosis, valve occlusion, prosthetic valve endocarditis, and peripheral thromboembolism. Two patients died suddenly in the late postoperative period. These deaths are assumed to be valve-related. For patients surviving the operative period, the linearized incidence of mortality related to valvular complications was 1.9 ± 0.5/100 patient-years.

Patient Survival

Long-term actuarial survival curves for the 176 operative survivors are shown in Figure 2. At 5 years the estimated survival rates were 70 ± 7.4% for AVR, 67 ± 6.0% for MVR, and 66 ± 9.7% for multiple-valve replacement. In patients surviving the operative period linearized mortality rate for AVR, MVR, and multiple-valve replacement was 4.4/100 patient-years, 6.3/100 patient-years, and 3.2/100 patient-years, respectively. The linearized rate for all surviving patients was 4.6 ± 1.6/100 patient-years. Overall, patients undergoing aortic valve replacement had significantly better survival, up to 48 months, than those undergoing mitral or multiple-valve replacement ($p = 0.0208$, Lee-Desu statistic). Rates of freedom from death or other complications are presented in Table 4.

Valve-Related Complications

THROMBOEMBOLISM. Thirteen thromboembolic episodes were observed, of which five were fatal (Table 3), for a mortality index of 38.5%. Ten of 13 events occurred within

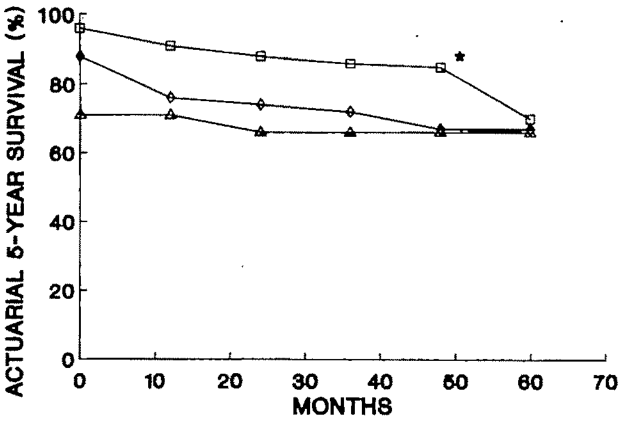

Fig 2. Actuarial 5-year curves for patients surviving the operative period. Survival for patients receiving isolated aortic valves (open square) *was significantly better, up to 48 months (*p = 0.0208), than for patients receiving mitral* (open diamond) *or multiple* (open triangle) *valves.*

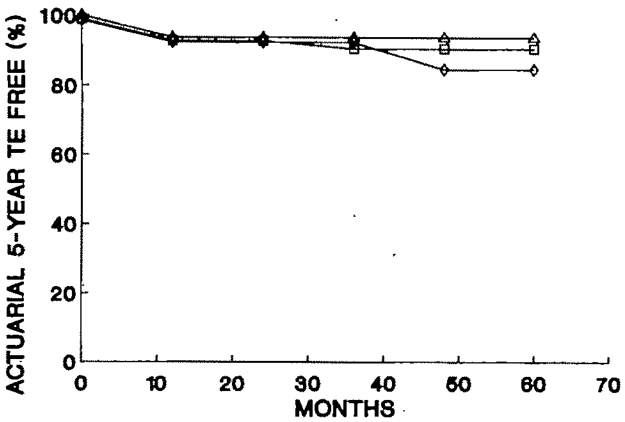

Fig 3. Actuarial 5-year curves for patients free of thromboembolic events. There were no significant differences between aortic (open square), *mitral* (open diamond) *or multiple* (open triangle) *valve groups.*

the first 2 years. Of the 8 patients suffering nonfatal events, 3 sustained permanent disability. The linearized incidence of thromboembolic events for all patients was 2.3 ± 0.3/100 patient-years. The overall actuarial 5-year freedom from thromboembolism was 90.0% ± 4.7% (Fig 3). There were no significant differences between valve groups.

VALVE THROMBOSIS. One fatal episode of thrombosis occurred in a patient receiving an aortic valve implant.

PROSTHETIC VALVE ENDOCARDITIS. Prosthetic valve endocarditis developed in 3 patients (1.5%) after five procedures, of which three required reoperation. The initial diagnosis was bacterial endocarditis in 2 of the 3 patients. Two of the patients (4 procedures) had received isolated aortic valves; the other patient underwent MVR. There was one death in this group (mortality index, 33.3%). The linearized incidence of this complication was 1.4/100 patient-years in the AVR group and 1.0/100 patient-years among patients receiving mitral valves. The overall 5-year actuarial freedom from endocarditis was 98.0% ± 2.3%. There were no significant differences between the groups.

Table 4. Actuarial 5-Year Freedom From Events[a]

Event	All Patients	AVR	MVR	DVR
Survival	67.7 ± 2.1	70.1 ± 7.4[b]	66.6 ± 6.0	66.3 ± 9.7
Thromboembolism	90.0 ± 4.7	91.7 ± 3.4	84.7 ± 8.0	93.8 ± 6.1
Valve thrombosis	99.6 ± 1.3	98.7 ± 1.3	100	100
Prosthetic valve endocarditis	98.0 ± 2.3	95.5 ± 2.2	98.5 ± 1.5	100
Paravalvular leak	94.5 ± 1.2	95.2 ± 2.3	93.1 ± 4.3	95.2 ± 4.6
Reoperation	92.1 ± 2.8	89.6 ± 3.3	91.6 ± 4.4	95.2 ± 4.6
Explantation	96.3 ± 3.2	94.2 ± 2.5	94.7 ± 3.9	100

[a] Values are shown as mean ± standard error of the mean. [b] $p = 0.0208$, AVR > MVR, DVR.

AVR = aortic valve; DVR = multiple valve; MVR = mitral valve.

NONSTRUCTURAL DYSFUNCTION. Paravalvular leakage that required reoperation was diagnosed in 4 patients with AVR, 3 with MVR, and 1 multiple-valve recipient. Four of the 8 patients had originally undergone urgent operation for subacute bacterial endocarditis. Reoperation was successful in 6 instances and fatal in 1 case each of AVR and multiple-valve replacement. The linearized rate of leak was 1.5 ± 0.1/100 patient-years overall with individual rates of 1.4/100 patient-years, 1.4/100 patient-years, and 1.6/100 patient-years for AVR, MVR, and DVR respectively. There were no significant differences in the incidence of paravalvular leak between groups.

One patient undergoing elective AVR suffered a sudden cardiac arrest perioperatively. Autopsy revealed suture material impinging on the valve orifice causing the disc to jam in the closed position.

There were no instances of death due to anticoagulant-induced hemorrhage.

REOPERATION. Fourteen patients (6.9%) required reoperation of which 13 procedures were performed within the first 12 months (Table 5). The most common indication was correction of paravalvular leak (8 patients). The most frequent valve replaced was in the aortic position (64%). There were three deaths after reoperation for a mortality index of 21.4%. Linearized incidence rates were 3.0/100 patient-years, 1.9/100 patient-years and 1.6/100 patient-years for AVR, MVR, and DVR patients respectively. The overall 5-year freedom from reoperation was 92.2% ± 2.9%. There were no significant differences between valves. Seven MH valves were removed and replaced with other prostheses (7/231 valves; 3.0%). Five aortic valves and two mitral valves were explanted. Three valves were removed for prosthetic valve endocarditis, 1 for paravalvular leak, and 3 for other causes. The linearized incidence of valve explantation was 1.4 ± 0.5/100 patient-

Table 5. Incidence of Reoperation

Cause of Reoperation	MVR	AVR	DVR	Total
Prosthetic valve endocarditis	1	2 (1)[a]		3 (1)
Paravalvular leak	3	4 (1)	1 (1)	8 (2)
Other		3		3
Total	4	9 (2)	1 (1)	14 (3)

[a] Numbers in parentheses are deaths.

AVR = aortic valve; DVR = multiple valve; MVR = mitral valve.

years. Again, no significant differences between patient groups were identified.

Comment

There is a considerable experience with the MH valve worldwide but there are few published reports, particularly from North America. Between 1982 and 1988 the MH valve became our mechanical valve of choice and this review details our experience.

Our failure to match the low frequencies of complications reported in some series [4, 10] likely reflects a difference in the severity of illness affecting our patients. Over half (53%) of the valves were implanted into patients who were in New York Heart Association class IV preoperatively and 96% of our patients were in NYHA class III or IV. This contrasts with other series involving the MH valve in which only 23% to 32% [5, 6] of patients were in class IV. Similarly the largest experience using the St. Jude Medical valve [10] involved patients of whom only 55% were NYHA class III or greater. Among our patients surviving the operative period, 75% improved substantially and are now in NYHA class I or II, which further attests to the value of valve replacement in these higher risk individuals.

The overall operative mortality in our series, 10.3%, is higher than literature reports of 5.7% to 9.6% [3, 4, 6]; however, 17 of the 21 operative deaths were in patients who were in NYHA class IV preoperatively. A second factor contributing to our mortality rate was the disproportionate number of operative deaths among the relatively small double-valve group. Over two-thirds of these patients were in NYHA class IV preoperatively and 25% underwent major associated procedures. The mortality rate for isolated valve procedures in our series averaged 3.4%. This figure compares favorably with other early experiences using the MH valve (2.7% to 5.1% [2, 6]) and with mortality rates reported for the St. Jude Medical valve [11, 12]. As in other series, perioperative heart failure was the leading cause of early mortality.

Patient survival, based on 5-year actuarial estimates, ranged from 66% to 70% among the three groups. The linearized rate of late mortality was 4.6 ± 1.6/100 patient-years. These figures are comparable with those of other published reports using the MH prosthesis [2, 5, 6] as well as with results available for other mechanical valves [10].

Death from valve-related complications affected 7.4% of our patients with an overall linearized rate of 1.9 ± 0.5/100 patient-years. This figure compares favorably with another experience using the MH valve with a rate of 1.6/100 patient-years [6]. In a large experience with this prosthesis under third-world conditions the valve-related mortality rate was 2.9/100 patient-years [5]. The valve-related death rate is, however, higher than the experience reported using the St. Jude Medical valve, with rates of between 0.86 [10] and 1.3 [13] per 100 patient-years in large series with up to 10-year follow-up.

When the MH valve was introduced in 1977, its presumed advantages over other tilting-disc and ball-valve prostheses included two-phase motion of the disc on the central guide rod to improve clearance when the valve was open and an increased opening angle to 75 degrees, thereby improving flow characteristics and reducing the pressure gradient across the valve. The improvement in flow was also thought to reduce the incidence of thromboembolism [1]. In vitro analysis [14] has demonstrated a reduced systolic pressure difference across the valve when compared with other tilting-disc models. Leakage volume, from blood regurgitating between the occluder and the valve ring when the valve is closed, is relatively high.

Thromboembolism and anticoagulant-induced hemorrhage are, in large part, complications of anticoagulation therapy. In our series the actuarial 5-year freedom from thromboembolism averaged 90% with individual rates of between 85% and 94% in the three valve groups. The valve's designers [4], who have the largest experience with the MH prosthesis, and others [6] report similar figures (84% to 93%). These data compare favorably with those for the St. Jude Medical valve, which is currently the most popular mechanical prosthesis in clinical use. The actuarial freedom from thromboembolism for this valve averaged 88% overall in a large series with 10-year follow-up [10]. The mortality index for thromboembolic events in our series was 38.5% compared with reports of 10% to 15% [5, 6] with the same valve. Problems of follow-up in the third-world population study [5] may be an important factor as 13% of their patients were lost to follow-up as compared with 5% in our series. A second potentially distorting influence would be the high numbers (16% [6] and 34% [5]) of patients suffering sudden or cause-unknown deaths compared with 7% in our series. These deaths are generally assumed to be caused by arrhythmias and thromboembolism. We found no instances of anticoagulant-induced hemorrhage severe enough to warrant transfusion or to cause death. This complication contributes between 0.5 and 1.2 events/100 patient-years in other studies and is a significant cause of mortality with rates between 21% and 36% [4, 6, 10]. Taken together, the linearized incidence of complications due to anticoagulation therapy in our patients was only 2.3/100 patient-years, which compares favorably with other experience using MH (3.3/100 patient-years [6]) and St. Jude Medical valves (2.5/100 patient-years [10]).

Prosthetic valve endocarditis was a complication after five procedures and affected 3 patients (1.5%). This figure is, however, skewed by a single patient who accounts for three of the five incidents. The indication for operation in

2 of the 3 patients was treatment of subacute bacterial endocarditis. Four of the five procedures involved isolated aortic valve implants for a linearized incidence of 1.4/100 patient-years in this group. One patient died of prosthetic valve endocarditis, giving a mortality index of 33%, comparable with published values of 29% [10] and 44% [5]. Mortality rates of 100% have been reported [13] and appear to be independent of type of prosthesis used [15]. In a large series of 1,104 patients with MH valves, the reported incidence was 0.9% [4]; others have recorded figures between 0.6% and 1.3% [6, 10].

Paravalvular leak affected 3.9% of our patients, a figure that, again, is higher than published elsewhere. Half of the patients had been operated on for subacute bacterial endocarditis with friable tissue and periannular abscesses noted at the initial procedure. There were no significant differences between the incidence in each of our three groups but leaks were more common when simple vertical sutures rather than pledgetted mattress sutures were used to secure the valve. The incidence of paravalvular leak in other series ranges from 0.8% to 2.3% [3–5, 10]. As a consequence of the high rates of prosthetic valve endocarditis and paravalvular leak, the incidence of reoperation in this series is greater than that reported by other authors [3–5, 10].

Mechanical obstruction was the cause of death in 1 patient after elective AVR. The patient suffered a cardiac arrest after a sudden rise in pulmonary pressures and a fall in systemic blood pressure. Attempts at resuscitation failed. At autopsy, suture material was found to be jammed between the disc and the valve orifice causing it to occlude. The valve had been secured with pledgetted 2-0 suture. Two other perioperative deaths are suspicious for valve impingement. Both patients had undergone MVR and each experienced sudden loss of cardiac output not related to size of valve used. Emergency reexploration revealed no obvious mechanical problem although both valves were rotated 90 degrees with no further difficulties experienced. Both patients died of cerebral ischemic complications secondary to the cardiac arrests. Extrinsic obstruction of the MH valve has been reported after MVR [16]; reexploration showed papillary muscle or suture material jamming the valve closed. The potential for impingement exists for all single-leaflet tilting-disc valves as a portion of the disc passes below the plane of the orifice during systole. Proper orientation of this type of valve may be important particularly in the mitral position when the posterior leaflet is preserved.

The MH valve has demonstrated itself to be a durable valve with acceptable levels of valve-related complications. There are two recent reports of large series with 10-year follow-up in which comparisons between the MH valve [4] and the St. Jude Medical device [10] can be made. These two are also the most commonly implanted mechanical protheses currently available. Rates for each of the complications described above were virtually identical between the two valves. In another recent study in which patients were randomized to receive one or the other of the two valves, comparison of rest and exercise hemodynamics demonstrated no advantage of one prosthesis over the other [17]. Clearly neither valve can claim superiority.

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Giant Chest Wall Tumor Resulting From Tissue Reaction to Foreign Bodies

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Three patients are reported in whom chest wall tumors developed 19 to 28 years after thoracoplasty and increased in size with time. The tumors could be removed operatively with good results. All tumors were composed of hematoma and necrotic material, and in all cases they revealed foreign bodies microscopically. Diagnosis and possible etiological factors are discussed.

(Ann Thorac Surg 1990;50:754–6)

Many surgical materials can induce foreign-body reactions, and rarely granuloma may develop [1]. We present the cases of 3 patients with unusually huge primary chest wall tumors that possibly resulted from foreign-body reactions to thoracoplasty.

Patient 1 was a 50-year-old man who had undergone left thoracoplasty for pulmonary tuberculosis at the age of 21 years. Some 19 years later and 10 years before admis-

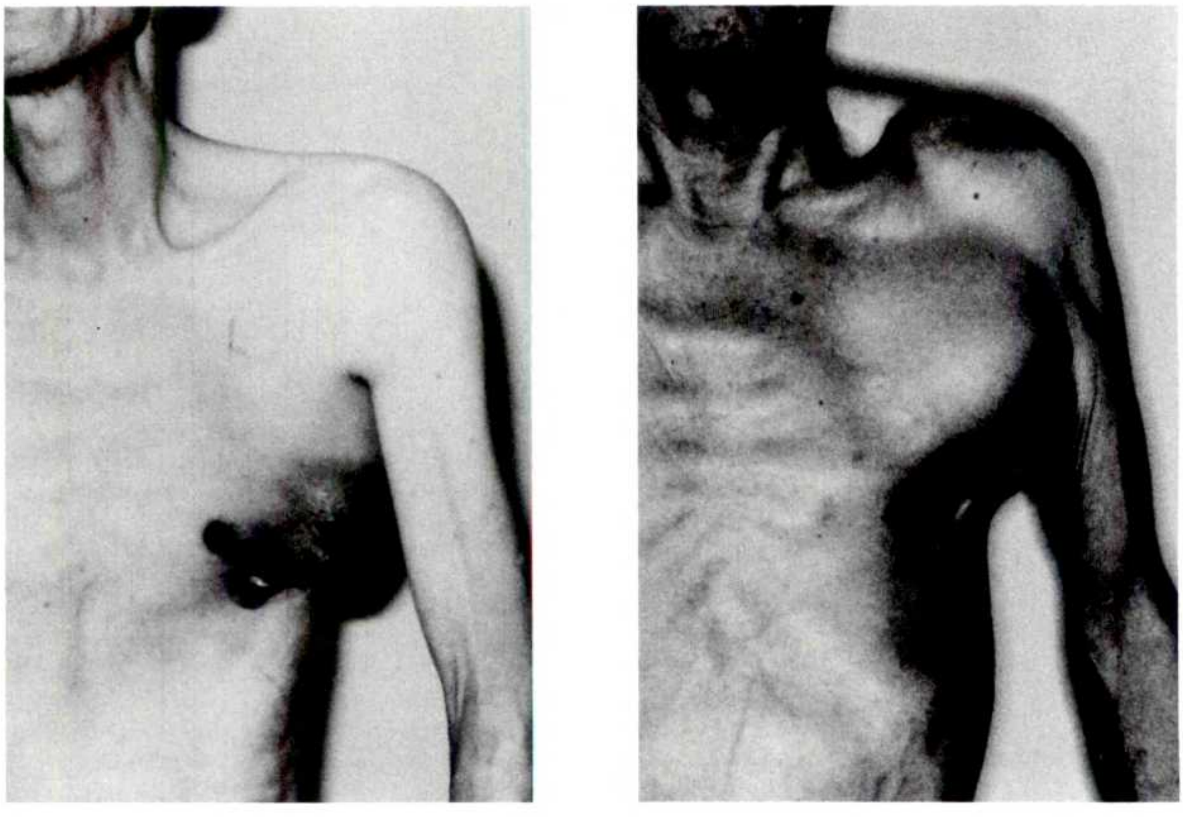

Fig 1. Huge mass on chest wall of patient 1 (A) and patient 2 (B). The skin of patient 1 is partly erosive.

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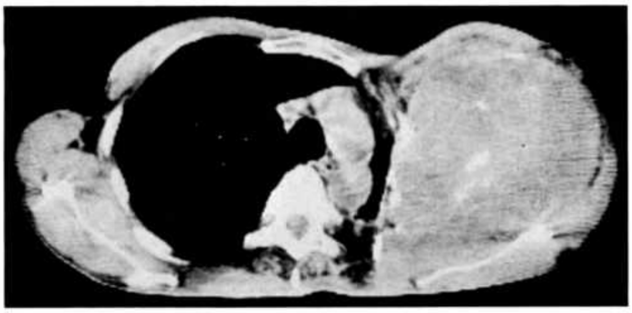

Fig 2. Computed tomographic scan of chest of patient 1, showing huge chest wall tumor compressing the bony chest wall and scapula.

sion, he noted a firm mass the size of a golf ball at the site of thoracoplasty. The mass increased in size with time until he had to be admitted (Fig 1A). Computed tomographic scan demonstrated a 12 × 10-cm, nonhomogenous mass (Fig 2). Biopsy was not helpful except to confirm presence of blood. At operation the tumor was not adherent to surrounding structures and was excised completely without thoracotomy. The mass measured 12 × 10 × 20 cm, weighed 1,513 g, and was composed of hematoma and necrotic tissue (Fig 3). Microscopically, capillary hyperplasia at the margin of the tumor, giant cells of foreign-body type, and foreign substances of unknown nature were observed.

Patient 2 was a 61-year-old man who had undergone left thoracoplasty for pulmonary tuberculosis at the age of 31 years. Twenty-eight years later and 2 years before admission, a firm, nontender mass at the site of thoracoplasty developed and enlarged during the subsequent interval (Fig 1B). Computed tomographic scan on admission revealed a 15 × 13-cm, nonhomogenous mass. At operation the tumor had no obvious vascular supply and was resected completely with relative ease. The excised specimen measured 15 × 13 × 9 cm, weighed 680 g, and consisted of hematoma and necrotic tissue. Meticulous multiple microscopic sections revealed the presence of textile fibers and capillary hyperplasia (Fig 4).

Patient 3 was a 63-year-old woman who had undergone right thoracoplasty for pulmonary tuberculosis at the age of 40 years. Twenty-three years later she noticed a slightly tender mass at the site of thoracoplasty. Two months later she was admitted to the hospital, where the lesion was excised. Computed tomographic scan demonstrated a 16 × 11-cm, nonhomogenous mass. The resected specimen measured 16 × 11 × 16 cm and weighed 780 g. It consisted of hematoma and necrotic tissue. Microscopically, capillary hyperplasia, foreign materials of unknown nature, and giant cells of foreign-body type were observed.

Comment

These 3 cases constitute a clinical and pathological entity; all patients had undergone thoracoplasty many years before the onset and the lesions all occurred at the operated sites. The gross and microscopic appearance of all tumors were strikingly similar.

Many kinds of foreign substances including surgical materials are capable of producing focal foreign-body reactions, in which lymphoid cells, plasma cells, and giant cells are present. The presence of giant cells in particular gives these reactions the appearance of granuloma [1]. As another cellular reaction to foreign materials, prominent capillaries may be pointed out [2]. Our 3 patients all revealed foreign bodies and capillary hyperplasia; no epithelioid cells, which are characteristic of tuberculosis, were observed. The possibility exists that the foreign material in patients 1 and 3 was paraffin. It is well known that foreign-body reactions after injection of paraffin occur as induration of subcutaneous tissue, and the interval between the time of injection and the development of induration can be many years [1].

From these findings, foreign materials such as gauze

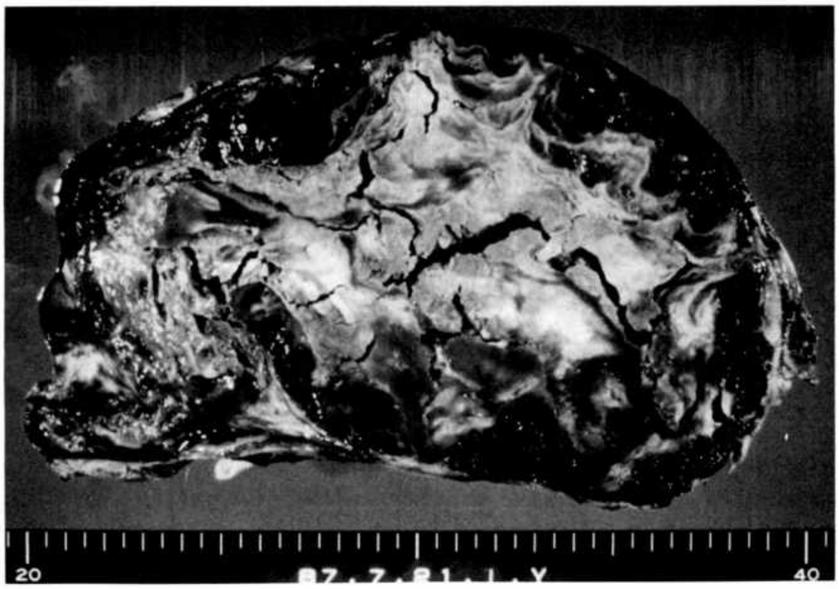

Fig 3. Cross-section of the tumor of patient 1.

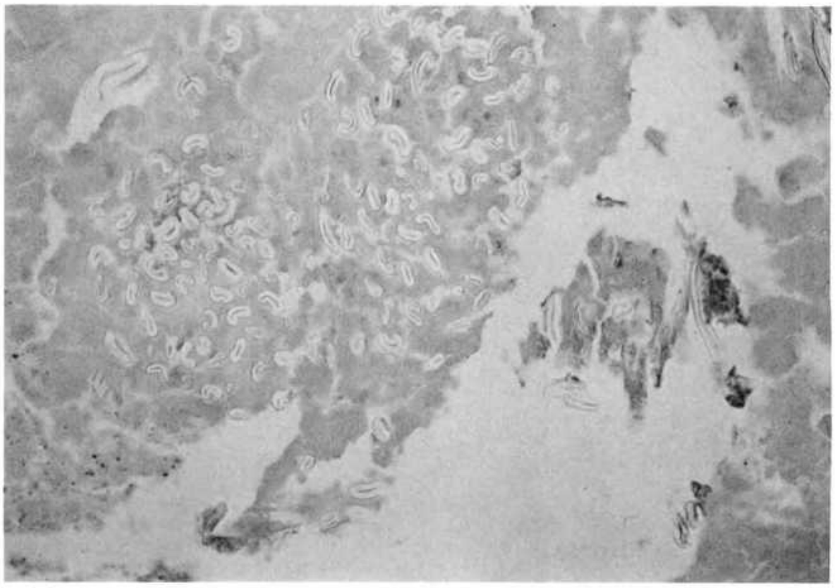

Fig 4. Many remnants of actual textile fibers (patient 2; hematoxylin and eosin, ×50 before 10% reduction).

and paraffin, which had been implanted during thoracoplasties, probably induced both granulomatous and capillary plastic reaction in our patients. At onset, granulomatous reaction might be prominent, at least in patients 1 and 2, because both tumors were firm and much smaller in size when they were noticed. Secondary, coincident capillary hyperplasia possibly caused repeated minor ruptures with time, so the tumors increased in size and older hematoma subsequently changed into organized tissue. This pathogenesis explains why all tumors were composed of hematoma and necrotic tissue on gross observation. The possibility exists that foreign-body granulomas are misdiagnosed when foreign materials cannot be recognized [3]. To detect foreign substances is essential, and polarizing microscopic sections may be more helpful to detect foreign bodies [3, 4]. In fact, the pathologists at first suspected hemangioma, which was in fact capillary hyperplasia, in patients 1 and 2. In our 3 patients, multiple microscopic sections, which revealed foreign bodies and foreign body reactions, and similar clinical features were helpful in making the proper diagnosis.

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Air Contamination in Open Heart Surgery With Disposable Coveralls, Gowns, and Drapes

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The effect of a polypropylene coverall, replacing shirt and trousers, combined with sterile laminated gowns and drapes compared with an all-cotton system was studied in regard to the dispersion of bacteria and particles in a conventionally ventilated operating theater. The operations carried out were open heart procedures in 30 adult patients. Blood agar sedimentation plates were placed in the operative, anesthesia, and perfusion areas. The mean sedimentation values during 1 hour after the start of operation were as follows in the laminate group: 63 colony-forming units (cfu)/m^2 in the operative area; 77 cfu/m^2 in the anesthesia area; and 143 cfu/m^2 in the perfusion area. The corresponding figures in the cotton group were 350 cfu/m^2, 364 cfu/m^2, and 437 cfu/m^2, respectively ($p < 0.0002$). At the beginning of the operation, the mean values noted for colony-forming units in the air at the operative site were 8.0 cfu/m^3 in the laminate group and 31 cfu/m^3 in the cotton group. One hour later, the values were 10 cfu/m^3 and 22 cfu/m^3, respectively ($p < 0.0002$). At the end of the operation, the number of particles 5 μm or larger in the air at the operative site was 278/m^3 in the laminate group and 592/m^3 in the cotton group. It is concluded that the use of a polypropylene coverall and laminated gowns and drapes significantly reduces the particle and bacterial contamination of the air and the bacterial sedimentation during cardiac operations.

(Ann Thorac Surg 1990;50:757–61)

Postoperative infections after open heart operations are associated with substantial morbidity and mortality [1, 2]. The most serious postoperative infection is prosthetic valve endocarditis, which has a mortality rate of up to 50% [1], and the gravest form of infection in coronary artery bypass procedures is a deep sternal wound infection associated with mediastinitis, which has a mortality rate of 10% to 20% [2–4]. Considering these serious risks, the development of prophylactic measures is of importance.

Although in some cases infection can result from postoperative contamination, intraoperative contamination is more obvious. In clean operations, where contact contamination is negligible, contamination of the wound is directly or indirectly due to air contamination [5, 6]. In cardiac operations, there are various possible routes of air contamination [7, 8]. In an experimental study in dogs [9], the oxygenator and cardiotomy reservoir were found to be contaminated mainly by aspiration of contaminated wound fluid. It has been found that aspiration of contaminated air into the extracorporeal circulation can cause bacteremia and thus poses a risk for postoperative infection [10].

The fact that airborne contamination can cause postoperative infection has been proved in orthopedic operations [11]. In cardiac procedures, where the operative area is large, the operating time is long, and the personnel are numerous, it can be assumed that clean air is of even greater importance. The present study was undertaken to evaluate the efficacy of the polypropylene coverall and laminated impermeable gowns and drapes compared with the traditional circulating all-cotton system in reducing airborne contamination in open heart operations.

Material and Methods

The operations chosen for the study were elective coronary artery bypass procedures carried out on 30 adult patients. The mean age of the patients was 51 years (range, 47 to 68 years). Preoperative whole-body wash with chlorhexidine gluconate soap was performed three times in all cases. The involved area was treated three times with 80% ethyl alcohol and 0.5% chlorhexidine solution.

The operations were done through a median sternotomy incision. Standard cardiopulmonary bypass with a bubble oxygenator was used. Mild general hypothermia and cold chemical cardioplegia were used as myocardial protection. The operative procedures were standardized as far as possible and were generally carried out by the same surgical team, consisting of three surgeons, two nurses, one anesthesiologist, one anesthesiology nurse, one perfusionist, and one perfusionist technician. In addition, there was one nurse taking samples. The operations were performed in the same conventionally ventilated operating theater with 20 changes of air per hour. The procedures included in the study were always the first in the morning. All patients were randomly allocated to either material system.

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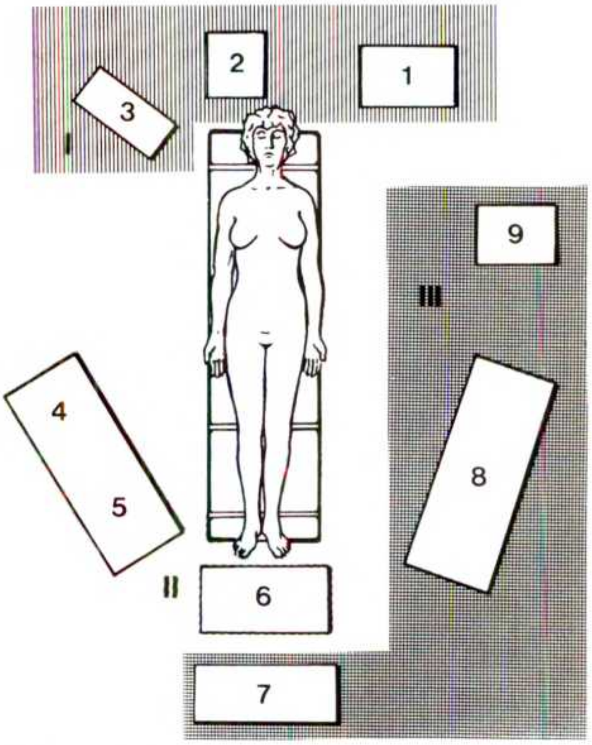

Fig 1. Layout of the operating theater and position of the sedimentation plates: anesthesia area (I) with anesthesia table (1), ventilator (2), and Sartorius filter sampler (3); operative area (II) with sterile instrument tables (4–6); and perfusion area (III) with diathermy (7), oxygenator and pumps for cardiopulmonary bypass (8), and defibrillator (9).

Clothing

Two different systems were used for patient draping and staff clothing. In the laminate group (15 patients), the patient was covered with plastic foil–laminated impermeable drapes with self-adhesive edges. All personnel working in the operating theater wore polypropylene coveralls, and the operating team used nonwoven operating gowns reinforced with plastic film in the front and lower parts of the sleeves (Mölnlycke Health Care AB, S-43581, Mölnlycke, Sweden).

In the cotton group (15 patients), the patient was covered with cotton drapes. The personnel in the operating theater wore cotton shirts, trousers, and conventional cotton operating gowns.

The patient draping was supplemented in every instance with incision foil (Steridrape; 3M, St. Paul, MN).

Bacteriological Air Sampling Studies

At each operation, nine sedimentation plates, 9 cm in diameter, with 2% horse blood nutrient agar were placed at different points 1 m above the floor in the operating theater (Fig 1): three in the operative area, three in the anesthesia area, and three in the perfusion area. The three sedimentation plates in each area were counted as one. The plates were exposed when the incision was made and left open for 1 hour. They were immediately incubated at 37°C for 18 hours and then at room temperature for 24 hours.

To assess bacteriological contamination, volumetric air samples were taken at the time of incision and 1 hour later 30 cm above the sternal wound using a Sartorius filter sampler (Sartorius GmbH, Göttingen, FRG) with a capacity of 30 L/min and a sampling time of 10 minutes. The filters, 5 cm in diameter with a pore size of 3 μm, were made of gelatin and were immediately transferred to blood agar plates for incubation aerobically for 48 hours at 37°C. The numbers of colonies were counted and were then typed using conventional clinical bacteriological techniques.

Particle Air Sampling

Three and a half hours after the start of the operation, particles 5 μm or greater were counted in 0.027 m^3 (1 ft^3) of air 30 cm above the wound by means of a Climet 0208A particle analyzer (Climet Instrument Co, Redlands, CA).

Statistical Analysis

The results obtained for the two material systems were compared using the Wilcoxon test.

Results

The sedimentation rates of aerobic bacteria in the three areas during 1 hour from the beginning of the operation are presented in Table 1. For both material systems, the air sedimentation rate in absolute numbers was higher in the perfusion area compared with the other two areas, though the difference was not significant. There was, however, a significant difference between the two material groups ($p < 0.0002$). The types of bacteria found are shown in Table 2. *Staphylococcus epidermidis* was the most common, but *Staphylococcus aureus* was also cultured from the sedimentation plates in two cases in the cotton group.

The influence of barrier material on the volumetric quantity of colony-forming units (cfu) is shown in Table 3. The fourfold difference between the two groups decreased but was still significant after 1 hour ($p < 0.0002$). In Table 4, the same phenomenon is demonstrated at different levels, ie, less than or equal to 10, less than or

Table 1. Air Sedimentation in the Operating Theater for 1 Hour From the Beginning of Operation[a,b]

Place of Samples	Laminate (cfu/m^2)	Cotton (cfu/m^2)
Operative area (n = 15 × 3)	63 (0–262)	350 (105–734)
Anesthesia area (n = 15 × 3)	77 (0–210)	364 (105–787)
Perfusion area (n = 15 × 3)	143 (0–367)	437 (0–1416)

[a] Data are shown as the arithmetic mean with range in parentheses.
[b] The difference between the sedimentation rates of the two material systems is significant ($p < 0.0002$).

Table 2. Bacteriological Findings on Sedimentation Plates

	No. of Operations	
Bacteria Isolated	Laminate (n = 15)	Cotton (n = 15)
S epidermidis alone	12	10
S epidermidis + *S aureus*	0	2
S epidermidis + other gram-positive cocci	0	2
S epidermidis + other bacteria	2	1
No growth	1	0

S = *Staphylococcus.*

Table 4. Number of Operations at Different Levels of Air Contamination

No. of Colony-forming Units per Cubic Meter	Laminate		Cotton	
	Start of Operation	+60 Minutes	Start of Operation	+60 Minutes
0	3	3	1	0
≤10	10	9	3	2
≤50	15	15	12	12
>50	0	0	3	3

equal to 50, and greater than 50 cfu/m^3, of air cleanliness. In three cases in the laminate group the air count was 0, both at the beginning of the operation and 60 minutes later. The corresponding figures in the cotton group were 1 and 0, respectively.

With the use of laminate, a level of 10 cfu/m^3 or less was noted at the start of the operation in 10 of the 15 patients and in 9 of the 15 after 1 hour. When cotton was used, the corresponding figures were 3 and 2, respectively. In no patient in the laminate group did the count exceed 50 cfu/m^3, whereas among the 15 cases in the cotton group, this was true for 3 operations at the start and 3 after 1 hour. No difference between the starting value and the value after 1 hour was noted in either group.

The number of particles 5 μm or larger 3.5 hours after the start of the operation was calculated. The particle count was 278/m^3 in the laminate group and 592/m^3 in the cotton group. The difference was significant ($p < 0.0002$).

Comment

In the technologically advanced fields of surgery, development is most rapid in cardiovascular surgery. This is true for both quantity and complexity. Postoperative infection is a serious risk in both prosthetic valve and coronary bypass grafting procedures. The correlation between wound cleanliness and the risk of infection has been proved both in vitro and in actual operations [11]. Measures of asepsis are aimed at preventing microorganisms from reaching the wound area. Whyte and coworkers [5] have shown that bacteria partly sediment from the air into the operative area and partly are transferred to the wound area through direct contact. Cardiovascular procedures involve a large sedimentation area, a long sedimentation time, and an abundant generation of airborne organisms because of the large number of personnel present. Therefore, in cardiovascular as well as in orthopedic operations, a barrier technique to reduce air contamination could be an effective measure of asepsis.

Table 3. Air Contamination at Operative Site[a]

Time of Sample	Laminate (cfu/m^3)	Cotton (cfu/m^3)
Incision	8 (0–26)	31 (0–100)
+ 60 minutes	10 (0–26)	22 (7–53)

[a] Data are shown as the arithmetic mean with range in parentheses.

A direct correlation between air contamination and infection rate has been demonstrated in surgical procedures requiring prostheses [5]. In orthopedic surgery, various ventilation systems have been developed to maintain ultraclean air conditions during long operations [11, 12]. These have been shown to give the highest level of air cleanliness when used in combination with ventilated gowns made of closely woven cotton. These systems, however, are limited in extent and inflexible and therefore are not suited for complex cardiac operations. Hence, it is of interest to study the possibility of reducing air contamination with the help of suitably designed working and barrier clothes.

These clothes have to be accepted by the individuals for whom they are intended and should have sufficient barrier properties. A garment of this kind has been tested in laboratory conditions [13] and in surgical situations [14]. In earlier studies [15, 16], some effects favoring nonwoven procedure-bound products have been demonstrated. With the system described in this study, however, it has been possible to attain a further reduction of air contamination. The minimum air contamination level for orthopedic operations, less than or equal to 10 cfu/m^3 of air, was achieved. The air contamination monitored in this study with a gelatin filter sampler is presented in Table 3. The counts were 8 and 31 cfu/m^3 at the beginning of the operation and 10 and 22 cfu/m^3 at 1 hour, respectively, for the two material systems used. The differences were highly significant ($p < 0.0002$).

The results in favor of laminate are well in accordance with the findings of Bergman and colleagues [14], even considering that the number of staff present during open heart operations is greater than during orthopedic procedures. In the present study, the number of personnel in the operating theater was ten.

There seems to be a lesser difference between the two systems as assessed in the perfusion area in comparison with areas close to the wound. A plausible explanation is that there is more physical activity in the perfusion area than around the operating table. This shows the importance of reducing movement as much as possible irrespective of the method used in controlling air contamination.

A greater difference in air contamination rates is observed initially rather than 1 hour later on comparing the laminate and cotton groups. This is due to the fact that whereas the counts for the laminate group remained unchanged, the counts for the cotton group decreased.

It has been well established that under normal conditions, air counts are highest at the beginning of the procedure because of the physical activity connected with the preparations for an operation [16]. The overall laminate system seems to be capable of maintaining a constant and lower count during this period. The capability of the impermeable laminate system to maintain lower levels of air contamination is demonstrated in Table 4, which shows the higher number of operations with lower counts.

The particulate contamination observed in this study may be derived from skin scales from personnel or patient, fibers from the materials used, or, more likely, both sources. Particles measuring 5 μm or greater represent the fraction of quickly sedimenting particles, and the results therefore ought to correlate with those from the sedimentation plates. This is of interest for two reasons. First, more direct methods of assessing the bacteriological contamination are required. Sampling with a slit sampler, eg, is relatively cumbersome. Second, the bacteriological techniques demand incubation of the samples for 24 or 48 hours. On-line systems are superior in the evaluation of methods reducing contamination. With a lowered total particulate air contamination, the sedimentation of foreign bodies into the wound also diminishes, and this may affect the infection rate, provided that the infection dose can be assumed to be low.

The material must be accepted by the personnel. It is important that a clothing system be comfortable. In our study, the benefit to the patient motivated the staff to use the barrier clothing tested.

The design of this study does not allow conclusions concerning the extent to which the various components of the laminate system contributed to the effects observed. However, in a recent study [17], the disposable gowns alone appeared not to reduce air contamination in hip operations. Further, in a test chamber, the same low level of air contamination has been noted irrespective of whether a laminate or cotton gown was worn over coveralls, a finding suggesting an important role of the coverall in reducing air contamination [13].

It can be assumed that a low degree of air contamination is important in avoiding postoperative wound infections [6]. This study presents a method to achieve and maintain desirable conditions. Further studies are needed to evaluate air contamination during the entire cardiac surgical procedure and the degree of wound contamination at the end of the procedure.

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INVITED COMMENTARY

Doctor Verkkala and colleagues have performed a careful study of the amount of viable bacteria in air with two separate types of drapes, gowns, and coveralls. Their results indicate a statistical reduction in the amount of colony-forming units of bacteria in the first hour of elective coronary artery bypass procedures. However, similar bacteria (*S epidermidis*) were present in both groups, and these organisms are a relatively uncommon component of postoperative sternal wound infections. Therefore, the reduction in colony-forming units in air with the impermeable gowns used in this study may or may not influence the rate of postoperative infection.

In the United States, impermeable gowns and drapes are now almost universally used, and therefore the clinical benefit of reduced infection rates by changing from cotton drapes to impermeable drapes has probably been achieved. The ideal material for gowns with respect to comfort, cost, safety, and efficacy has not been found and continued evaluation should be encouraged.

This paper is interesting from the standpoint of infection control, and I am pleased to see interest in these problems in the thoracic surgical literature.

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Notice From the American Board of Thoracic Surgery

The American Board of Thoracic Surgery began its recertification process in 1984. Diplomates interested in participating in this examination should maintain a documented list of the operations they performed during the year prior to application for recertification. This practice review should consist of 1 year's consecutive major operative experiences. (If more than 100 cases occur in 1 year, only 100 need to be listed.) They should also keep a record of their attendance at approved postgraduate medical education activities for the 2 years prior to application. A minimum of 100 hours of approved CME activity is required.

In place of a cognitive examination, candidates for recertification will be required to complete both the general thoracic and cardiac portions of the SESATS IV syllabus (Self-Education/Self-Assessment in Thoracic Surgery). It is not necessary for candidates to purchase SESATS IV booklets prior to applying for recertification. SESATS IV booklets will be forwarded to candidates after their applications have been accepted.

Diplomates whose 10-year certificates will expire in 1993 may begin the recertification process in 1991. This new certificate will be dated 10 years from the time of expiration of the original certificate. Recertification is also open to any diplomate with an unlimited certificate and will in no way affect the validity of the original certificate.

The deadline for submission of applications is July 1, 1991. A recertification brochure outlining the rules and requirements for recertification in thoracic surgery is available upon request from the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Aortic Dissection With the Entrance Tear in Transverse Aorta: Analysis of 12 Autopsy Patients

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Clinical and autopsy findings are described in 12 patients who had fatal aortic dissection with the entrance tear in the transverse aorta. The 12 patients represent 7% of 182 autopsies of spontaneous aortic dissection studied by us. The ages of the 12 patients at death ranged from 37 to 87 years (mean, 67 years). Eight were men; 8 had a history of systemic hypertension, and 10 had hearts of increased weight. Diagnosis of aortic dissection was made during life in only 4 of the 12 patients. All 12 patients died of rupture of the false channel within 2 weeks of onset of signs or symptoms compatible with dissection. The direction of aortic dissection from the entrance tear was entirely retrograde in 4 patients, entirely anterograde in 4 patients, and in both directions in 4 patients. Hemopericardium occurred in the first group, left hemothorax in the second group, and either in the last group. Of the 8 patients in whom the ascending aorta was involved, the retrograde dissection in each extended to the aortic root, 6 had pulmonary adventitial hemorrhage, and 4 had involvement of the arch arteries by dissection. In the 4 patients with strictly anterograde dissection, none had dissection in the arch arteries. Thus, tear in the transverse aorta causes a dissection that is usually fatal, that often dissects retrogradely, and that may mimic dissection from a tear in ascending aorta. Aortic dissection from a tear in transverse aorta requires early operative intervention.

(Ann Thorac Surg 1990;50:762–6)

During the past 30 years, 182 patients with spontaneous (noniatrogenic) aortic dissection have been studied in the Pathology Branch, National Heart, Lung, and Blood Institute. The entrance tear was located in the transverse aorta in 12 (7%) of the 182 patients, and they are the subject of this report.

Material and Methods

The records of the Pathology Branch from 1959 through March 1990 were searched for cases of aortic dissection. A total of 194 cases were found and, after review of each record, 12 were excluded from further analysis because of an iatrogenic cause—the entrance tear occurred at the site of a previous aortotomy. Of the remaining 182 patients, the entrance tear was located in the ascending aorta in 127 (70%), descending thoracic aorta in 40 (22%), abdominal aorta in 2 (1%), and transverse aorta in 12 (7%). This report describes findings in the latter 12 patients. The transverse aorta is defined as that portion of thoracic aorta extending from an imaginary line corresponding to the beginning of the origin of the innominate artery to an imaginary line corresponding to the end of the origin of the left subclavian artery.

The hearts and aortas in all 12 patients were originally examined and classified by W.C.R., and the specimens from 9 patients were examined by C.S.R. and reexamined by W.C.R. Of the 12 patients, 1 was studied at autopsy in 1969; 7, from 1971 to 1980; and 4, from 1981 to 1990. The 12 cases were submitted from 10 different hospitals, one of which submitted 3 cases.

Results

The clinical and autopsy findings in each of the 12 patients are summarized in Table 1. Their ages ranged from 37 to 87 years (mean, 67 years); 8 were men and 4 were women. Systemic hypertension had been present by history in 8 patients (67%), and 10 patients (83%) had hearts of increased weight (>350 g in women and >400 g in men). Of the 12 patients, only 4 had the diagnosis of aortic dissection established during life. Of the 5 patients who had an aortogram performed, diagnosis of suspected aortic dissection was confirmed in 3.

All 12 patients died of rupture of the wall of the aortic false channel within 14 days of onset of symptoms or signs compatible with aortic dissection. One patient underwent operative therapy (patient 6, Table 1), but only after rupture of the false channel. Plication of the channel in the ascending aorta was performed after a prolonged period without blood pressure, and the patient died of hemorrhage in the operating room. In no patient did the aortic dissection heal.

The longitudinal extension of aortic dissection was only in a retrograde direction in 4 patients (33%) (Figs 1, 2), only in an anterograde direction in 4 (33%) (Figs 3, 4), and in both directions in 4 (33%). In all 4 patients in whom the

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Table 1. Clinical and Morphologic Characteristics of 12 Patients Who Had Aortic Dissection With an Entrance Tear in the Transverse Aorta

							Dissection		Dissection Involves:		Dissection in:				Bleeding Into:							
Patient No.	Age at Death (y)	Sex	SH	Aortogram	AD Diagnosed Clinically	Interval AD to Death (d)	R	A	DTA	AA	IA	LCCA	LSA	TC Stenosis	PS	LH	AD Behind Aortic Sinuses	AH PT	Stenosis PT	AAA	HW (g)	LV Scar
1	60	F	+	0	+	2	+	0	0	0	0	0	0	0	+	0	+	+	0	0	500	0
2	64	M	+	+	0	4	+	0	0	0	0	0	0	+	+	0	+	+	0	+	475	0
3	65	F	+	+	0	8	+	0	0	0	+	0	0	+	+	0	+	+	+	0	. . .	0
4	80	F	+	0	0	1	+	0	0	0	0	0	0	+	+	0	+	0	0	+	475	0
5	37	M	0	0	0	1	+	+	+	+	+	+	+	0	+	0	+	+	+	0	365	0
6	65	M	+	+	+	2[a]	+	+	+	+	0	+	0	0	+	0	+	+	+	0	430	0
7	67	M	0	+	+	14	+	+	+	+	+	0	0	0	0	+	+	+	0	0	420	+
8	67	M	+	0	0	6	+	+	+	+	0	0	0	0	0	+	+	0	0	0	650	0
9	53	M	0	0	0	4	0	+	+	+	0	0	0	0	0	+	0	0	0	0	820	0
10	75	M	+	+	+	6	0	+	+	+	0	0	0	0	0	+	0	0	0	0	440	+
11	78	M	+	0	0	4	0	+	+	+	0	0	0	0	0	+	0	0	0	+	600	0
12	87	F	0	0	0	1	0	+	+	+	0	0	0	+	0	+	0	0	0	+	370	0
Total or mean	67	M, 8 F, 4	8	5	4	≤14	8	8	8	8	3	2	1	4	6	6	8	6	3	4	M, 525 F, 448	2

[a] During hemorrhagic shock from aortic rupture, this patient underwent plication of the false channel but died at operation.

A = anterograde; AA = abdominal aorta; AAA = abdominal aortic aneurysm; AD = aortic dissection; AH = adventitial hemorrhage; DTA = descending thoracic aorta; F = female; HW = heart weight; IA = innominate artery; LCCA = left common carotid artery; LH = left hemithorax; LSA = left subclavian artery; LV = left ventricle; M = male; PS = pericardial sac; PT = pulmonary trunk; R = retrograde; SH = systemic hypertension by history; TC = true channel.

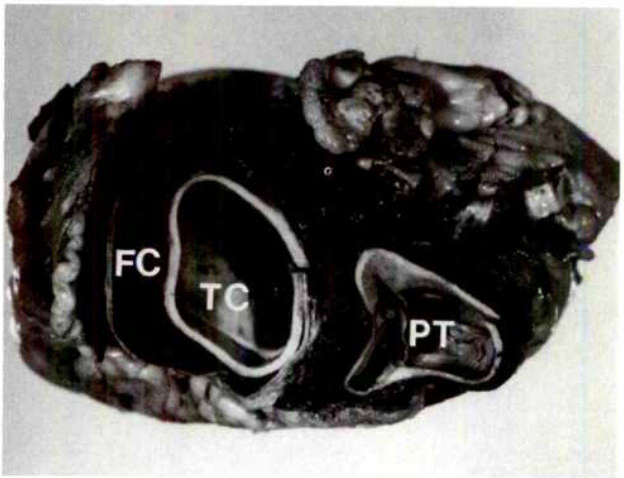

Fig 1. (Patient 1.) View of the heart and great arteries of a 60-year-old woman who died within 2 days of onset of symptoms and signs of aortic dissection. The entrance tear was located in the aortic arch and the direction of dissection was entirely retrograde. The aorta and pulmonary trunk (PT) *share adventitia and consequently share adventitial hemorrhage, which in this case was not severe enough to cause pulmonary arterial stenosis. Rupture of the aortic false channel* (FC) *caused hemopericardium and death.* (TC = *true channel.)*

dissection was retrograde only, rupture of the false channel occurred in ascending aorta producing hemopericardium. In all 4 patients in whom the dissection was anterograde only, rupture of the false channel occurred in descending thoracic aorta, producing left hemothorax. In the 4 patients in whom dissection occurred in both directions, hemopericardium occurred in 2 and left hemothorax in 2.

Of the 8 patients in whom retrograde dissection occurred, all had hemorrhage in the aortic wall behind the sinuses of Valsalva, and 6 had pulmonary adventitial hemorrhage, which occurs because the aorta and pulmonary trunk have a common adventitia and because the medial aortic dissection ruptures into the aortic media [1–3]. In 3 of these 6 patients, the pulmonary adventitial hemorrhage was severe enough to cause compression of the true lumen of the pulmonary trunk.

Of the 12 patients, 4 had involvement of 1 or more of the arch arteries by dissection: innominate artery in 3 patients; left common carotid artery in 2 patients, and left subclavian artery in 1. In the 4 patients in whom the dissection was anterograde only, none of the arch arteries were involved by dissection. Thrombus was present in the false channel in all 12 patients. The false channel aneurysm was large enough in 4 of the 12 patients to produce stenosis of the true channel by compression.

Comment

Analysis of the aforementioned 12 autopsy patients with aortic dissection from an entrance tear in transverse aorta disclosed that all patients died within 2 weeks, that the dissection often (8 patients) went retrogradely simulating dissection with the entrance tear in ascending aorta, that rupture caused hemopericardium as often as left hemothorax, and that healing of the dissection occurred in none. Aortic dissection with the entrance tear in the transverse aorta appears to have the highest acute fatality rate of any type of dissection, and its direction of extension is the most difficult to predict.

The transverse aorta is an uncommon site for the entrance tear in dissection. Hirst and associates [4], in their collective review of 505 previously published cases, found the transverse aorta to be the entrance site in 9% and the ascending aorta in 62%. Among our 182 autopsy

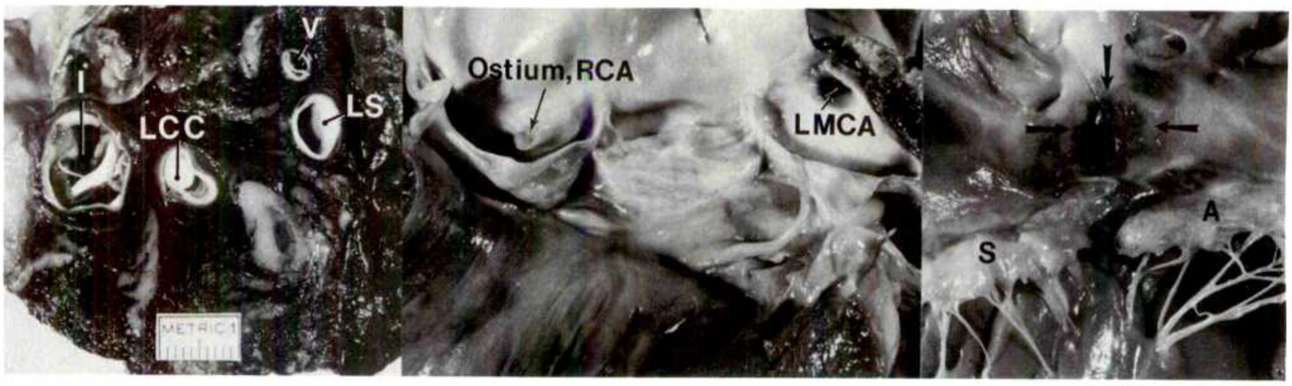

Fig 2. (Patient 5.) Aortic arch arteries (left), opened aortic valve (center), and opened right side of the heart (right) in an 80-year-old woman who had the entrance tear in the transverse aorta and an entirely retrograde dissection. The ascending aorta ruptured into the pericardial sac. (Left) View of innominate (I), *left common carotid* (LCC), *left subclavian* (LC), *and vertebrae* (V) *arteries from above showing dissection in each of these four arteries and hemorrhage into the surrounding tissues. (Center) The right coronary artery* (RCA) *has been completely severed by the retrograde dissection allowing inversion* (arrow) *of its proximal portion into the aortic sinus* (LMCA = *left main coronary artery.) (Right) This photograph shows portions of the right atrium and right ventricle and septal* (S) *and anterior* (A) *tricuspid valve leaflets. The arrows designate a hemorrhagic area in the right atrium—a consequence of the retrograde aortic dissection—in the area of the atrioventricular node. Such hemorrhage can lead to complete heart block in patients with aortic dissection.*

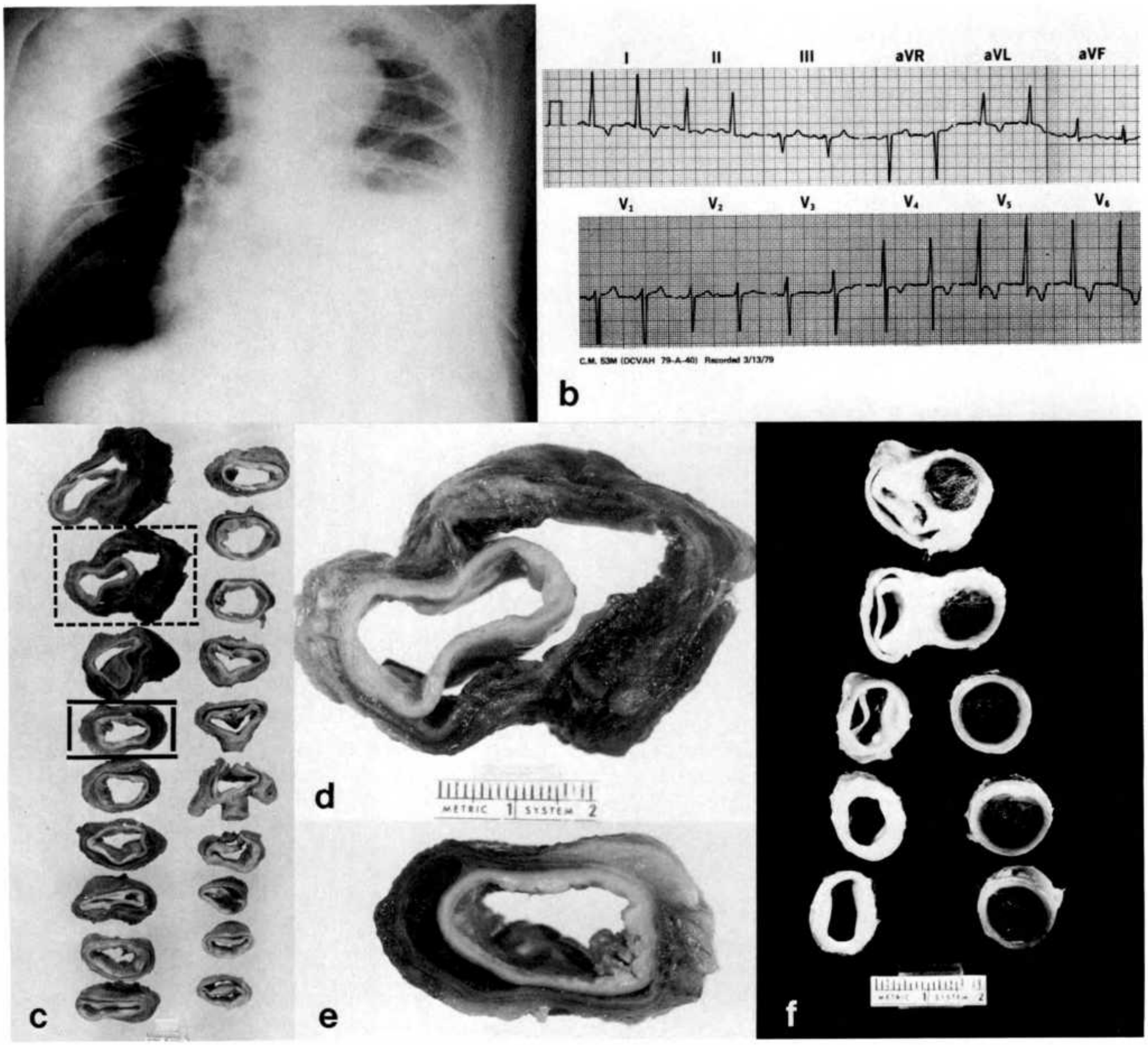

Fig 3. (Patient 9.) Chest radiograph (a), electrocardiogram (b), transverse sections of descending thoracic and abdominal aorta (c), close-up views of two sections from c (d, e), and transverse sections of common iliac arteries (f) in a 53-year-old man who had fatal hemorrhage into the left hemithorax. The dissection was entirely anterograde. The chest radiograph shows a large left pleural effusion. The electrocardiogram is consistent with left ventricular hypertrophy from the systemic hypertension. The transverse sections show a dilated false channel proximally and dissection without aneurysm distally. (d) and (e) are close-up views of the two transverse sections in brackets *in (c). The false channel in one common iliac artery is filled with thrombus to the extent that it obliterates entirely the false lumen in one artery.*

cases of spontaneous aortic dissection, the tear was located in the transverse aorta in 7% and in the ascending aorta in 70%. Miller [5] also has emphasized that an entrance tear in the transverse aorta is rare. Of 175 patients with aortic dissection who underwent operative therapy at Stanford over a 20-year period, 22 (13%) had entrance tears in the transverse aorta. In those 22 patients, dissection went retrogradely in 11 (50%) and anterogradely in 11 (50%). Similarly, of our 12 patients, the dissection went retrogradely in 8 and anterogradely in 8, including the 4 patients in whom the dissection went in both directions.

Proper therapy of aortic dissection when the entrance tear is located in transverse aorta has been controversial. Because operative therapy for this condition has yielded high mortality and morbidity rates, most patients with tears in transverse aorta have been treated by nonoperative means. The Stanford group [6, 7], however, has advocated operative therapy, with few exceptions, for aortic dissection irrespective of the site of the entrance tear. When the tear is located in transverse aorta, Miller [5] recommended resection of the entrance tear, even though a transverse aortic arch operation has a higher operative risk than an operation limited to ascending or

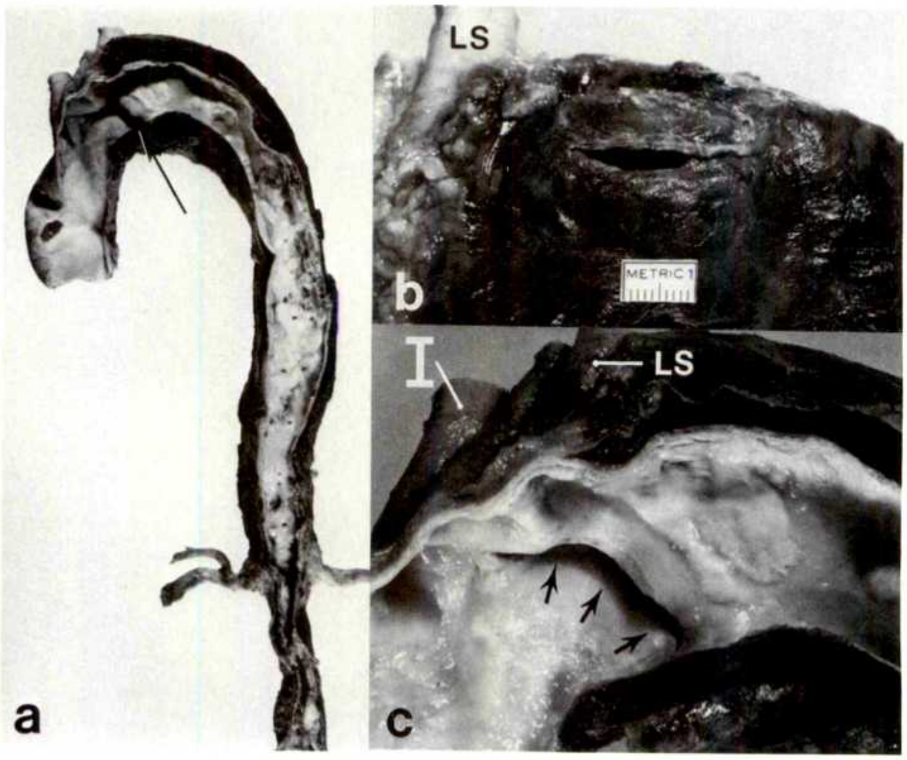

Fig 4. (Patient 10.) Opened aorta (a), site of external aortic perforation (b), and interior of transverse aorta (c) in a 75-year-old man who had an anterograde dissection with fatal rupture into the left hemithorax. The arrows *in (a) and (c) point to the tear in the transverse aorta, a close-up of which is seen in (c). (I = innominate artery;* LS *= left subclavian artery.)*

descending aorta [8–14]. We support the view that patients with aortic dissection from an entrance tear in transverse aorta should undergo immediate operation, including resection of the entrance tear.

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Unusual Opening of Coronary Sinus in Atrioventricular Septal Defects

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The coronary sinus is an important landmark for the position of the atrial component of the atrioventricular conduction axis and, thus, assumes special relevance to the surgeon in the operating room. We describe here 2 patients with atrioventricular septal defect characterized by an unusual termination of the coronary sinus within the left atrium. We discuss the potential importance of this finding to the disposition and surgical avoidance of the conduction tissues.

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The coronary sinus occupies a crucial position within the septal structures of the heart. Although its right atrial orifice is not strictly septal, it occupies the atrioventricular (AV) groove at the crux of the heart and is directly contiguous with both the inferior rim of the oval fossa and the muscular AV septum. As such, its mouth forms the base of the normally structured triangle of Koch and is, therefore, an important landmark to the AV node. In some circumstances, the coronary sinus is said to be congenitally absent [1, 2]. This has been described either when the course of the coronary sinus within the left AV groove is unroofed, the "orifice" persisting as a right atrial landmark, or when the entire structure is lacking, as typically seen in hearts with isomerism of the right atrial appendages ("asplenia").

We have now encountered two hearts, both with deficient AV septation, in which the atrial and ventricular septal structures were malaligned. The coronary sinus terminated in the left atrium, and there was no evidence of its usual "orifice" within the atrial septum. Indeed, the entire part of the septum that usually joins the oval fossa to the crux and encircles the mouth of the coronary sinus (the sinus septum) was lacking. Such an anomaly of the coronary sinus was not included within the detailed categorization of Mantini and associates [3]. This paper reviews the anatomy of this crucial part of the septal structure, highlighting its absence in the two abnormal hearts, and discusses the importance of this finding for surgical avoidance of the AV conduction axis.

Case Histories

Patient 1

A previously asymptomatic 14-year-old boy was referred for evaluation of exertional dyspnea. Pertinent physical findings included a subxiphoid ventricular impulse, wide split S_2 with a normal P_2, a grade 3/6 long, harsh, systolic murmur maximal at the upper left sternal border, and a grade 2/4 early diastolic rumble at the lower left sternal border. An electrocardiogram revealed first-degree heart block, biatrial enlargement, −70-degree QRS frontal axis, and right bundle-branch block; a chest radiograph showed cardiomegaly and increased pulmonary vasculature. Examination by cross-sectional echocardiography (Fig 1) demonstrated marked malalignment of the atrial and ventricular septa with a large interatrial communication secondary to a deficiency of the AV septum; the coronary sinus was visualized in the left atrium. No "cleft" was noted in the left AV valve. Cardiac catheterization findings included a pulmonary/systemic blood flow ratio of 4, moderate right ventricular systolic hypertension (52 mm Hg), a large left to right atrial shunt confirmed by angiography and oximetry, and absence of left AV valve regurgitation.

At the time of operation, the patient was noted to have an unusual AV septal defect. When the right atrium was opened, a large interatrial communication could be seen beneath the lower part of the atrial septum. The orifice of the coronary sinus was found in the posterior wall of the left atrium (Fig 2A) rather than in its anticipated location (Fig 2B). Inspection of the atrioventricular valves showed that they were attached to a common atrioventricular junction and were separated by a tongue of leaflet tissue into right and left orifices. The tongue itself was attached along the crest of the ventricular septum, but did not show any evidence of division into superior and inferior components, as expected in a typical "ostium primum" defect. Instead, the left AV valve had two leaflets located in septal and mural location, with the plane of the commissure between them being parallel to the ventricular septum (Fig 3). The overall junctional morphology was that of an AV septal defect, although the left AV valve had two rather than its anticipated three leaflets. Other than the absence of the sinus septum the remainder of the atrial septum was intact except for a probe-patent foramen

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During the course of this investigation, R.H.A. was on study leave from the National Heart and Lung Institute, London, United Kingdom.

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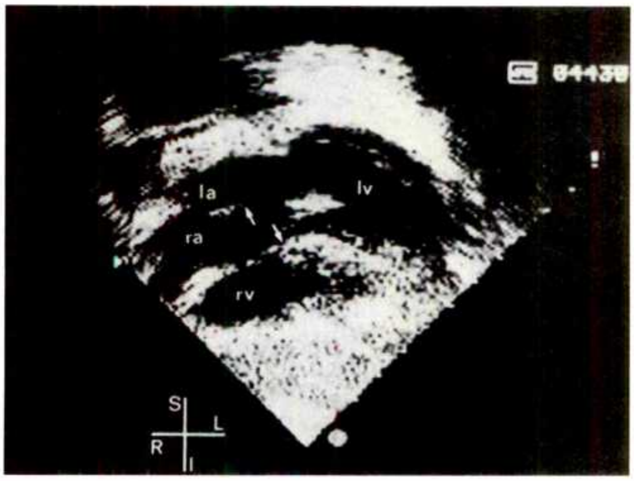

Fig 1. The cross-sectional echocardiogram from patient 1 shows a deficiency at the anticipated site of the atrioventricular septum with marked malalignment of the atrial and ventricular septal structures (arrow). *(*la = *left atrium;* lv = *left ventricle;* ra = *right atrium;* rv = *right ventricle.)*

ovale. The defect was closed with pericardium in a fashion such that sutures were taken in the large valvar leaflet tissue along the crest of the septum. Contrary to our usual practice of using the right AV valve tissue to anchor the patch, the patulous, intact left AV valve tissue was used here. Thus, the suture line ran on the left ventricular side of the defect and extended onto the posterior left atrial wall so that venous flow from the coronary sinus was directed into the right atrium. The superior suture line was brought round on the atrial wall so that the oval fossa was transferred over to the left atrium, thereby closing the probe-patent defect. The patient was weaned from bypass without difficulty and made an uneventful postoperative recovery.

Patient 2

An asymptomatic 6-year-old girl with Down's syndrome and an AV septal defect diagnosed at birth (and status post ligation of an arterial duct during infancy) was admitted for elective surgical repair. The cardiac examination was noteworthy for a subxiphoid ventricular impulse, wide but variably split S_2 with a normal P_2, a grade 2/6 systolic ejection murmur at the upper left sternal border, a grade 2/6 long, harsh apical systolic murmur, and an early grade 2/4 diastolic murmur at the lower left sternal border. Electrocardiographic abnormalities included a QRS frontal axis of −90 degrees and right bundle-branch block; radiographic abnormalities included increased pulmonary vascular markings and mild cardiomegaly. Cross-sectional echocardiographic examination (Fig 4) revealed a deficiency of the AV septum with marked malalignment of the atrial and ventricular septa and a "cleft" in the bridging leaflets of the left AV valve; the coronary sinus entry appeared to be into the left atrium. Pulsed Doppler echocardiographic mapping demonstrated a large left to right atrial shunt and mild left AV valve regurgitation. Pertinent cardiac catheterization findings were mild right ventricular systolic hypertension (46 mm Hg), a pulmonary/systemic blood flow ratio of 2.5, a large oximetric and angiographic left to right atrial shunt, and mild left AV valve regurgitation.

At operation she was found to have an AV septal defect. The large interatrial communication was located in its usual place, but the coronary sinus was displaced posteriorly, opening directly into the left atrium (Fig 5). Noteworthy is the fact that, as in the previous patient, no thebesian valve was associated with the orifice. The morphology of the AV valve was as expected with the anticipated three leaflets guarding the left AV junction (Fig 6; cf, Fig 3). Because of this we chose to anchor our sutures along the septal crest in the right AV valve tissue. Thus, the pericardial patch was inserted so that the

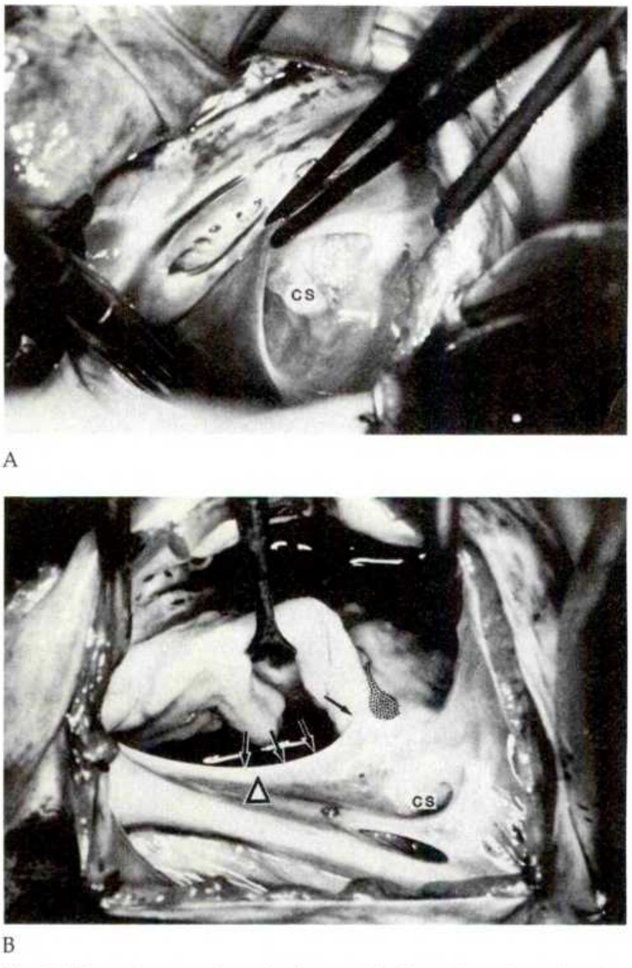

Fig 2. These views are through the opened right atrium in patient 1. (A) shows the sucker tip beneath the lower edge of the atrial septum. The forceps are picking up the orifice of the coronary sinus (cs), which opens in the posterior aspect of the left atrium. Crystalloid cardioplegic solution is emanating from the sinus. No thebesian valve is seen. Compare this arrangement with the usual arrangement found in atrioventricular septal defect (B). The lower edge of the atrial septum (arrows) *forms the sinus septum enclosing the mouth of the coronary sinus. Note the regular location of the triangle of Koch (△). The conduction tissue* (hatched area)*, however, is displaced to the crux of the heart.*

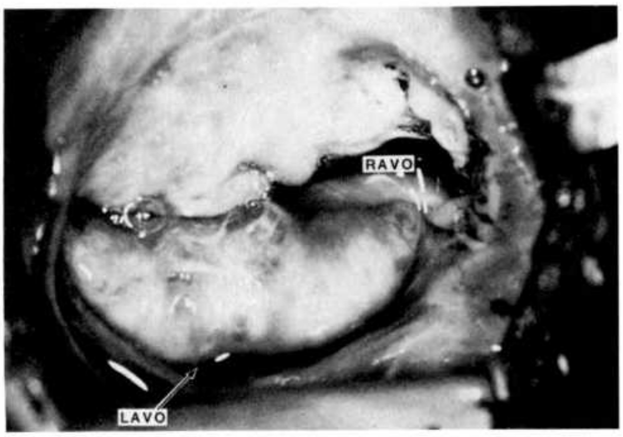

Fig 3. This view shows the detailed morphology of the leaflet tissue separating the right and left atrioventricular orifices in patient 1. Unlike the usual situation in an atrioventricular septal defect, the tissue shows no evidence of separation into superior and inferior components (absence of so-called cleft). (LAVO *and* RAVO = *left and right atrioventricular orifices.)*

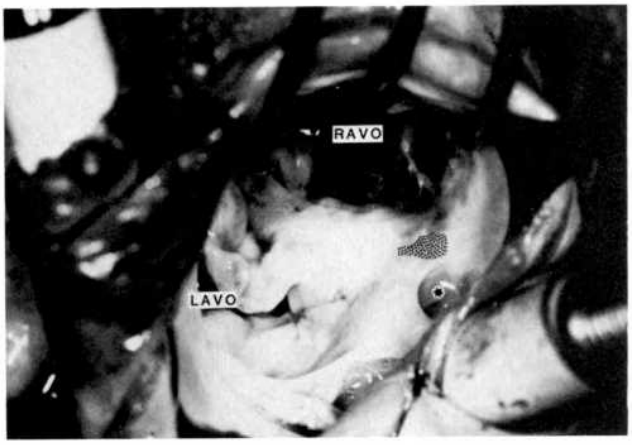

Fig 5. This view through the right atrium shows the junctional anatomy in patient 2. There are separate atrioventricular orifices (RAVO, LAVO) *but the coronary sinus (*) opens into the posterior aspect of the left atrium. The anticipated site of the conduction axis is shown by the* dotted area. *Note the absence of the thebesian valve.*

coronary sinus continued to drain into the left side of the heart (Fig 7). The patient had an uneventful postoperative course.

Comment

The most detailed study of anomalous formation of the coronary sinus was made by Mantini and colleagues in 1966 [3]. As far as we can judge, they did not encounter lesions comparable with those presently described. The essence of the malformation seen in our 2 patients is that the sinus itself is normally formed and runs within the left AV groove, but then terminates within the left atrium. The entire component of atrial wall and septal surface that normally connects the oval fossa to the crux (Fig 8), the so-called sinus septum, is lacking in the hearts we have described. The major importance of the deranged anatomy is its influence on the landmarks of the AV conduction axis.

The apex of the triangle of Koch serves, in the normal heart, as the guide to the point of penetration of the atrioventricular axis of conduction tissue. In patients with AV septal defect, the structure of the triangle is deranged by the lack of AV septal structures, yet an analogue of the triangle can be found in the inferior rim of the atrial septum. This triangle, however, does not contain the AV node. Instead, the node is deviated posteriorly, being contained within that part of the septal surface that still

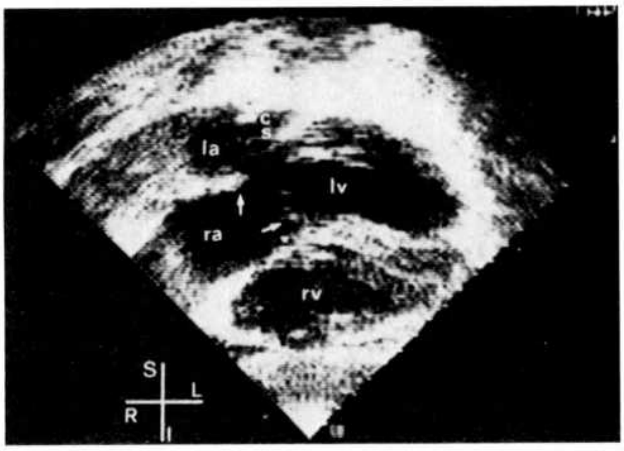

Fig 4. A cross-sectional echocardiogram in four-chamber projection in patient 2 shows an atrioventricular septal defect with marked malalignment of the atrial and ventricular septal structures (arrows). *The coronary sinus* (cs) *is seen in the left atrioventricular groove.* (la = *left atrium;* lv = *left ventricle;* ra = *right atrium;* rv = *right ventricle.)*

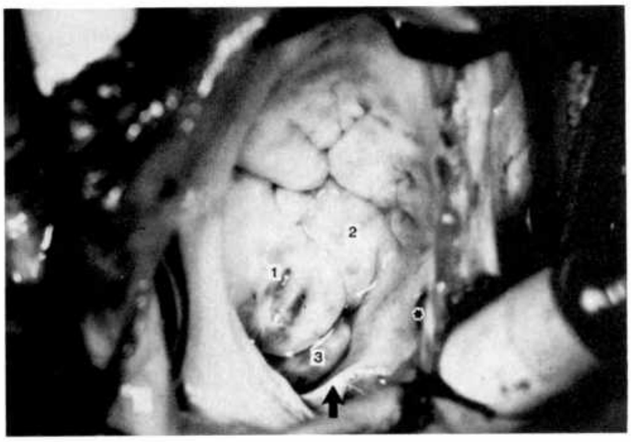

Fig 6. This view shows the arrangement of the leaflets of the right and left atrioventricular valves. The left valve has three leaflets (1, 2, 3). Note also the malalignment between the lower edge of the atrial septum (arrow) *and the ventricular septum beneath the bridging leaflets. The atrial septum fails to enclose the mouth of the coronary sinus (*).*

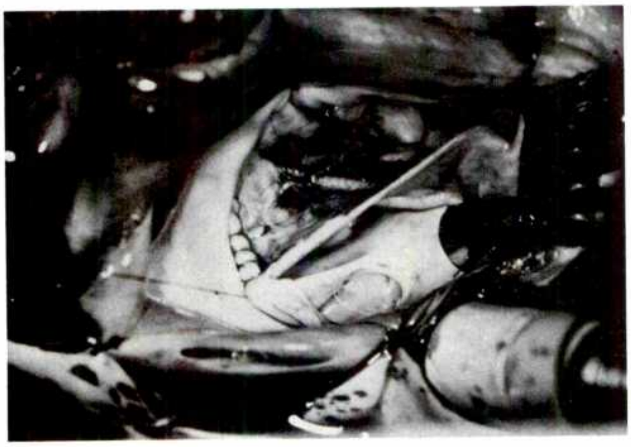

Fig 7. The patch is inserted and sutures placed in all but its postero-inferior margin. When this is closed, the coronary sinus, with the forceps in its orifice, will drain to the newly septated left atrium.

surrounds the termination of the coronary sinus (see Fig 2B). This landmark was totally lacking in the two hearts presently described because of the septal malalignment associated with the missing sinus septum. Consequently, the coronary sinus terminated within the left atrium.

This points to a general principle that still holds good in such situations [4]. The AV node in the presence of septal malalignment is found at the point where the ventricular septum meets with the AV junction. This is also the case in hearts with straddling of the tricuspid valve, where it is the ventricular septum that is deviated away from the crux [5, 6]. It is also the case in these two hearts, where it could be argued that the atrial septum is deviated away from the crux with a normal location of the ventricular septum. Be that as it may, the AV node is still present at the point where the ventricular septum reaches the AV junction. It must be said that, in our cases, this is a presumption, as we did not map electrically the location of the AV bundle. In neither patient, however, did atrioventricular rhythm disturbances develop after operative repair, which was based on the aforementioned principles.

Not directly related to the anomalies of the coronary sinus, it is, nevertheless, remarkable that, in our first patient, the left AV valve possessed only two leaflets and lacked the expected septal commissure (or "cleft") typically found in the setting of deficient AV septation. The orifice of the left valve was oriented parallel to the septum, rather than having a component at right angles to the septum, as is usually the case. Although we have never previously encountered hearts of this type, in which the bridging leaflets formed a confluent structure, there can be no doubt that the heart had all other anticipated features of an AV septal defect.

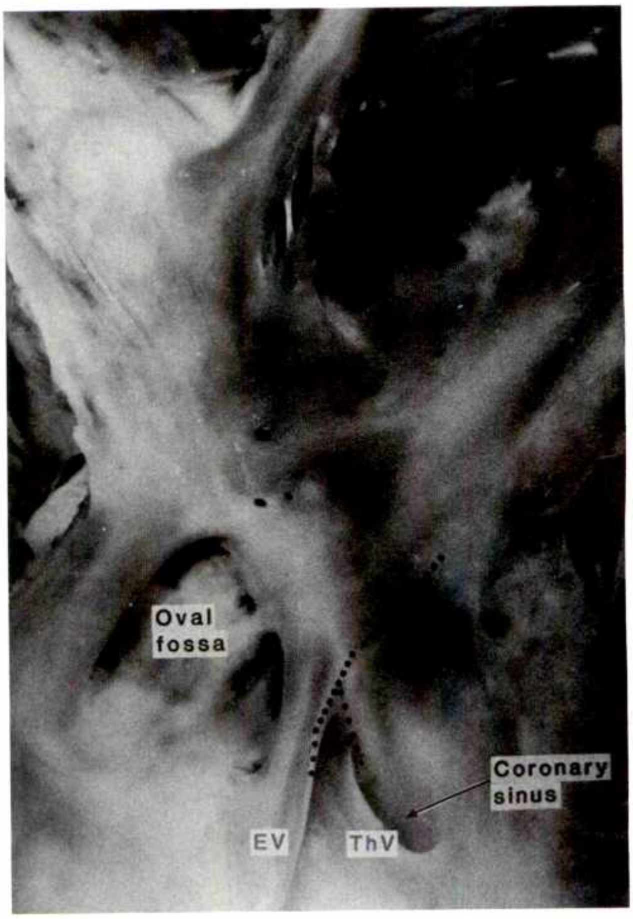

Fig 8. This view of the normal right atrium shows the sinus septum and the course of the tendon of Todaro (dotted line) *formed by fusion of the eustachian* (EV) *and thebesian* (ThV) *valves.*

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Myocardial Preservation Using Lidocaine Blood Cardioplegia

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Prevention of ventricular fibrillation after aortic unclamping using lidocaine hydrochloride as an additive to cold potassium blood cardioplegia was studied prospectively in 46 patients undergoing elective myocardial revascularization. Patients were similar with respect to age, ventricular function, severity of coronary artery disease, cross-clamp time, completeness of revascularization, frequency of internal thoracic artery grafting, systemic temperature at the time of cross-clamp removal, and mean infusate volume and temperature. Patients receiving lidocaine blood cardioplegia (group 1, 23 patients) had a significant reduction in the incidence of ventricular fibrillation (22% versus 74%; $p < 0.0005$) and in the mean number of cardioversion attempts required to defibrillate the heart (0.5 ± 1.3 versus 1.9 ± 0.97; $p < 0.0005$) after cross-clamp removal compared with controls (group 2, 23 patients). There were no differences between the two groups postoperatively with regard to cardiac enzyme release, hemodynamic measurements, or clinical outcome. Patients receiving lidocaine blood cardioplegia tended to have a lower incidence of new postoperative atrial fibrillation (9% versus 26%). Ventricular function was preserved equally in both groups. We conclude that lidocaine is a safe additive to potassium blood cardioplegia and significantly reduces the incidence of ventricular fibrillation after aortic unclamping.

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Prolonged ventricular fibrillation can adversely affect coronary blood flow and increase ventricular wall tension. Under normothermic conditions, this could contribute to myocardial ischemia and depress ventricular performance. Prevention of ventricular fibrillation is one aspect of the overall strategy for minimizing myocardial ischemia during open heart operations.

The period just after aortic declamping commonly is characterized by spontaneous ventricular fibrillation. In this setting, prolonged fibrillation could negatively influence ventricular function, particularly in the immediate postbypass period. Lidocaine hydrochloride is well known to prevent ventricular ectopy in patients after a myocardial infarction. There is evidence to suggest that lidocaine can also exert a protective effect on ischemic, reperfused myocardium [1]. The purpose of this study was to assess the safety of lidocaine as a blood cardioplegia additive and determine its effectiveness in decreasing the incidence of ventricular fibrillation after aortic declamping in patients undergoing elective myocardial revascularization.

Material and Methods

From January 1, 1988, to December 31, 1988, 46 patients undergoing elective coronary artery bypass grafting prospectively alternately received cold blood cardioplegia through the aortic root with (group 1, 23 patients) or without (group 2, 23 patients) the addition of lidocaine. The protocol was approved by the institutional review board at St. Louis University Group Hospitals on November 15, 1988 (IRB No. 5355).

Data recorded included age, sex, history of a prior transmural myocardial infarction, number of vessels bypassed, frequency of internal thoracic artery grafting, and left ventricular end-diastolic pressure. Regional wall motion abnormality was measured by left ventricular score as defined by the Coronary Artery Surgery Study [2]. Major coronary artery stenosis was defined as occlusion of 50% or more of the left main trunk and luminal diameter stenosis of 70% or more of other arteries in any angiographic plane.

Anesthesia management included endotracheal intubation and maintenance with fentanyl (20 to 50 mg/kg), pancuronium bromide (0.1 mg/kg), and enflurane. Patients were ventilated with 100% oxygen. Catheters were placed in a radial artery, and a balloon-tip catheter (Opticath P7110; Oxymetric, Inc, Mountain View, CA) was positioned in the pulmonary artery for hemodynamic measurement and systemic venous oxygen saturation.

Cardiopulmonary bypass was established using ascending aortic and dual-stage right atrial cannulation employing moderate systemic hypothermia (30°C nasopharyngeal temperature) and hemodilution (hematocrit, 0.20 to 0.25). The extracorporeal circuit contained a membrane oxygenator (SciMed model I-3500-2A; SciMed

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Life Systems, Inc, Minneapolis, MN) primed with 1,800 mL of crystalloid and 500 mL of 5% albumin.

The composition of the cardioplegic infusate and its delivery were identical in both groups except for the addition of lidocaine (0.1 mg/mL), administered to group 1 patients only. The lidocaine hydrochloride did not contain preservatives or epinephrine. The cardioplegic infusate consisted of 4:1 dilution of blood with 0.9% normal saline solution, which contained 20 mEq/L of sodium bicarbonate. The initial dose was 10 mL/kg and contained 14 mEq/L of KCl. Reinfusions (5 mL/kg; 6 mEq/L of KCl) were performed at 20-minute intervals into the aortic root and through each completed saphenous vein graft. All patients received cardioplegia through the aortic root using a DLP catheter (model 23009; DLP, Walker, MI) at an aortic root pressure of 80 to 100 mm Hg.

Topical myocardial cooling with Ringer's lactate slush with a cardiac insulation pad for phrenic nerve protection was employed in all patients. A left ventricular vent was not used. Proximal anastomoses were performed using a partially occlusive aortic clamp, and distal anastomoses were constructed with the aorta cross-clamped. The volume and temperature of the cardioplegic solution and the time required for infusion were recorded as well as the interval to achieve cardiac arrest and the ventricular septal temperature (Shiley myocardial temperature probe DPM; Shiley Inc, Irvine, CA).

Recorded operative variables included cardiopulmonary bypass and cross-clamp times, the number of trials necessary to separate the patient from the extracorporeal circuit, and the number of electrical shocks required to achieve ventricular defibrillation. The need for epicardial pacing or the administration of additional antiarrhythmic drugs to treat ventricular ectopy after cross-clamp removal were also noted. No patient was on a regimen of antiarrhythmic therapy preoperatively.

Blood samples for assessment of lactate dehydrogenase isoenzymes 1 and 2 (LDH_1 and LDH_2) were obtained on postoperative days 1 and 2. A ratio between LDH_1 and LDH_2 of 1.0 or greater was considered indicative of myocardial injury. Measurements of the myocardial-specific isoenzyme of creatine kinase (CK-MB) were obtained before aortic cross-clamping and at 1, 2, 3, 4, 8, 12, and 24 hours after unclamping. A myocardial fraction greater than 50 U/L was considered diagnostic of myocardial injury. The serum potassium level immediately after cross-clamp removal was also recorded.

Hemodynamic measurements were obtained at 0.5, 1, 2, 3, 4, 8, 12, and 24 hours after cross-clamp removal. Measurements recorded included systemic, right atrial, pulmonary artery, and pulmonary capillary wedge pressures and cardiac output (thermodilution technique). Using standard formulas, values for cardiac index and right ventricular and left ventricular stroke work indices were calculated at each interval.

Data were analyzed using the Statview II statistical software package (Brainpower, Inc, Calabasas, CA). Univariate analysis of discrete variables was performed using χ^2 analysis or Fisher's exact test where appropriate. Student's *t* test was employed for continuous variables. A *p* value of less than 0.05 was considered significant. Significant differences for left ventricular and right ventricular stroke work indices and creatine kinase curves were determined by two-factor balanced analysis of variance for repeated measures. Mean values are expressed as ± the standard deviation or standard error of the mean where indicated.

Table 1. Summary of Patient Data[a]

Variable	Group 1 (n = 23)	Group 2 (n = 23)	*p* Value
Age (y)	62	67	NS
Sex (male)	12	19	NS
Left ventricular score	7.6 ± 3.0	9.0 ± 2.8	NS
LVEDP	18.0 ± 5.9	16 ± 7.5	NS
No. of diseased vessels	2.7 ± 0.422	2.9 ± 0.288	NS
Left main disease	7	3	NS
No. of vessels bypassed	3.3 ± 0.93	3.7 ± 1.0	NS
Internal thoracic artery (%)	74	74	NS
Duration of CPB (min)	116.4 ± 31.5	115.0 ± 28	NS
Cross-clamp time (min)	72 ± 25.9	64.4 ± 21.2	NS

[a] Where applicable, data are shown as the mean ± the standard deviation.

CPB = cardiopulmonary bypass; LVEDP = left ventricular end-diastolic pressure; NS = not significant.

Results

Preoperative and intraoperative patient data are shown in Table 1. There were no differences between groups with respect to age, sex, left ventricular function, severity of coronary artery disease, completeness of revascularization, frequency of internal thoracic artery grafting, duration of extracorporeal circulation, or cross-clamp time.

The temperature of the infusate was 5°C in all patients, and the mean ventricular septal temperature achieved was not different between the groups (group 1, 12.2° ± 3.5°C, and group 2, 10.6° ± 3.0°C; p = not significant [NS]). Although a greater volume of cardioplegic solution was used in group 1, the difference was not significant (22.8 ± 7.2 mL/kg versus 21.2 ± 5.5 mL/kg; p = NS).

The time to achieve complete electrical arrest was nearly identical in both groups (group 1, 16.9 ± 21 seconds, and group 2, 17.5 ± 19 seconds; p = NS). In no patient did electromechanical activity return before cross-clamp removal or during intervals between cardioplegia infusions.

The extracellular serum potassium concentration at the time of cross-clamp removal was virtually identical in both groups (group 1, 4.2 mEq/L, and group 2, 4.3 mEq/L; p = NS), as was the systemic temperature (group 1, 30.2° ± 1.4°C, and group 2, 29.7° ± 1.1°C; p = NS). There was a significant reduction in the incidence of ventricular fibrillation in patients receiving lidocaine blood cardioplegia (group 1, 22%, and group 2, 74%; $p \le 0.0005$). The mean number of attempts required to defibrillate the heart also was decreased in patients receiving lidocaine blood cardioplegia (group 1, 0.5 ± 1.3, and group 2, 1.9 ± 0.97; $p \le 0.0005$). Of the 5 patients in the lidocaine group requiring defibrillation after cross-clamp removal, 1 patient had sustained an intraoperative myocardial infarction and required intravenous administration of lidocaine and procainamide hydrochloride to control ventricular ectopy in the postoperative period. The remaining 4 patients had systemic temperatures of 28°, 29°, 31°, and 32°C at the time of cross-clamp removal. Reperfusion may have introduced intramyocardial temperature gradients causing spontaneous ventricular fibrillation in these patients.

At the time of cross-clamp removal, the systemic temperature was nearly identical in patients whose hearts fibrillated (22 patients, 29.9° ± 1.4°C) compared with those whose hearts resumed normal electrical activity (24 patients, 30.0° ± 1.2°C; p = NS). Patients whose hearts fibrillated were compared with those who had resumption of normal electrical activity with regard to left ventricular score (9.0 ± 3 versus 7.6 ± 2.6), extent of coronary artery disease (2.8 ± 0.4 versus 2.8 ± 0.3 diseased vessels), completeness of revascularization (3.4 ± 1.1 versus 3.5 ± 0.8 vessels bypassed), and cross-clamp time (63 ± 21 seconds versus 74 ± 25 seconds). No significant differences were observed.

There were no operative deaths, and no patient required intraaortic balloon counterpulsation. Use of inotropic agents after termination of cardiopulmonary bypass was nearly identical in both groups (group 1, 26%, and group 2, 22%; p = NS), as was the number of trials required to separate the patient from the extracorporeal circuit (group 1, 1.0 ± 0.20, and group 2, 1.0 ± 0.21; p = NS).

Six patients in both groups required temporary epicardial pacing. No patient had permanent heart block. The frequency of new-onset postoperative atrial fibrillation was greater in the control group (9% versus 26%), but the difference was not significant.

No significant difference was noted between group 1 and group 2 with respect to the ratio of LDH_1 and LDH_2 levels measured in the first and second postoperative days. The CK-MB curve demonstrated peak enzyme levels at 2 hours after aortic unclamping in both groups (Fig 1). One patient in the lidocaine group sustained a perioperative transmural infarction as defined by new Q waves in the electrocardiogram and elevated levels of myocardial isoenzymes. Despite this infarct in a group 1 patient, a trend toward lesser enzyme release was noted in patients receiving lidocaine blood cardioplegia, although the difference was not significant.

There was a very early trend toward better left ventricular function in those patients not receiving lidocaine cardioplegia, but the difference reached significance only at 30 minutes after aortic unclamping (Fig 2). There was no significant difference thereafter when the curves were compared by analysis of variance. Right ventricular performance was well preserved in both cohorts with no significant differences noted at any time (Fig 3).

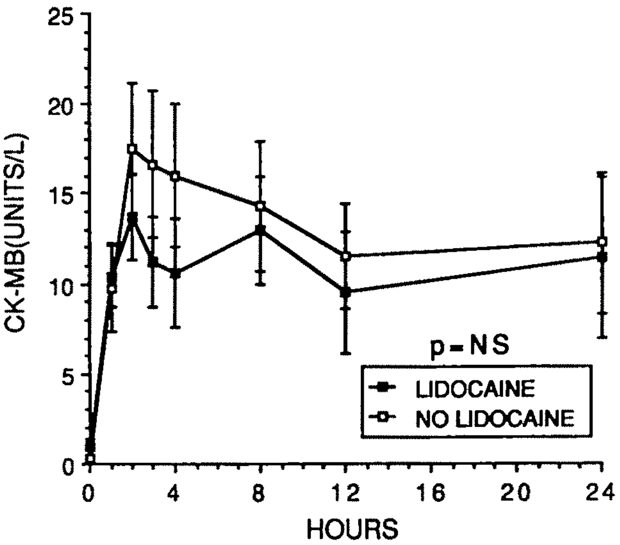

Fig 1. Comparison of myocardial enzyme release: systemic blood levels of the myocardial-specific isoenzyme of creatine kinase because (CK-MB) before and after aortic cross-clamping. Mean values are shown ± the standard error of the mean. (NS = *not significant.)*

Comment

Reperfusion injury after global ischemia is a complex and incompletely understood cellular and biochemical process. However, it has been well established that reperfusion injury is characterized by major changes in membrane permeability [3]. Reperfusion damage leads to activation of phospholipases, which results in damage to and disorganization of membrane ultrastructure. This membrane damage results in cellular efflux of potassium and magnesium, influx of sodium and water, and ultrastructural damage to and reduction in the functional capacity of mitochondria [4].

The beneficial effect seen with cardioplegia containing lidocaine may be due to its effects at the cellular level. Many local anesthetics have been shown to have a mem-

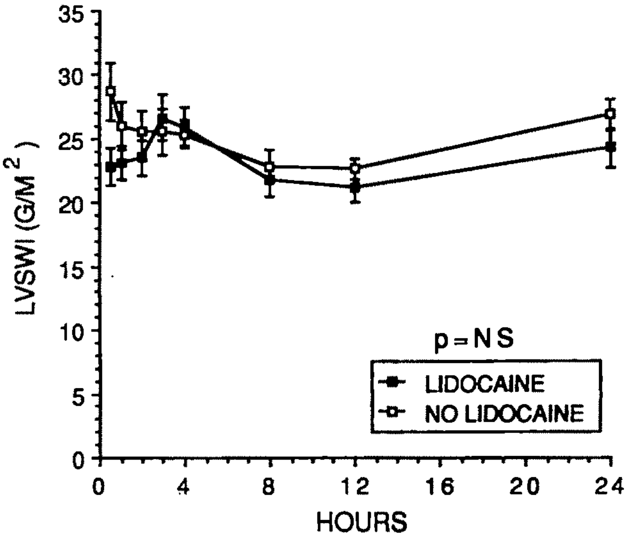

Fig 2. Left ventricular stroke work index (LVSWI) at intervals after aortic cross-clamp removal. Mean values are shown ± the standard error of the mean. (NS = *not significant.)*

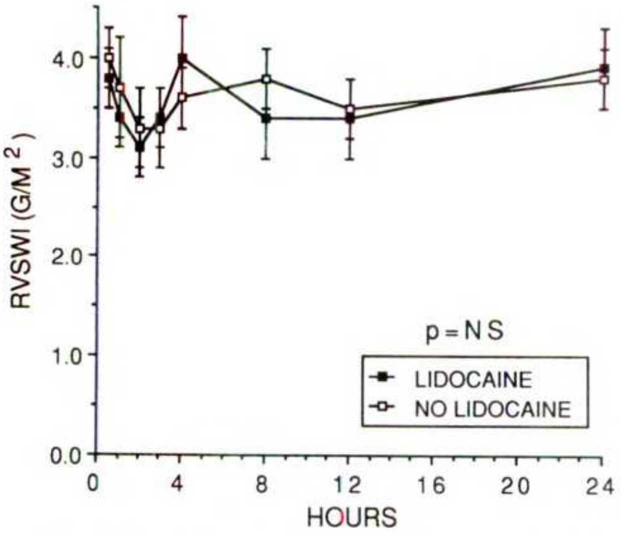

Fig 3. Right ventricular stroke work index (RVSWI) *at intervals after aortic cross-clamp removal. Mean values are shown ± the standard error of the mean.* (NS = *not significant.*)

brane-stabilizing effect [5]. Seppala and associates [6] demonstrated that dibucaine inhibited phospholipase A_2 activity and thereby helped to maintain membrane phospholipid integrity. Similarly, Seeman [7] demonstrated that lidocaine increased the viscosity of lipid bilayers and inhibited ionic flux across the mitochondrial membranes.

In the clinical setting, the physiological effects and ultrastructural changes associated with reperfusion can be demonstrated in the setting of acute myocardial infarction. Experimentally, Naseer and associates [8] demonstrated reduction in infarct size and improvement in mitochondrial respiratory function in animal hearts continuously perfused with lidocaine after temporary left anterior descending coronary artery occlusion with subsequent reperfusion. This beneficial effect of lidocaine was not associated with depression of left ventricular function.

The clinical reperfusion phenomenon most frequently seen by thoracic surgeons occurs after cross-clamp removal during the performance of coronary artery bypass grafting. In this setting, reperfusion of the heart after the global ischemia of cardioplegia-induced arrest is frequently characterized by the occurrence of ventricular fibrillation. Prolonged ventricular fibrillation, particularly under normothermic conditions, can cause subendocardial ischemia with depressed ventricular function in the immediate postbypass period. Although lidocaine is considered the drug of first choice for perioperative ventricular arrhythmias, most thoracic surgeons are unfamiliar with this drug as an additive to blood cardioplegic solutions for the prevention of ventricular fibrillation so often associated with reperfusion.

Blood cardioplegic solutions containing local anesthetics have not gained popularity in the United States. The addition of procainamide to cardioplegic solution has been suggested to result in two specific disadvantages [9]. First, procainamide has been associated with a depression of myocardial contractile function in the immediate postbypass period. Second, it was thought that procainamide prevented the rapid resumption of electrical activity after aortic unclamping.

In the experimental setting, delayed resumption of electrical activity has not been demonstrated with the use of lidocaine. Kyo and colleagues [10] infused up to 350 mg of lidocaine as an additive to crystalloid cardioplegia using an in vivo canine model of global myocardial ischemia. They observed no delay in the resumption of electrical activity during the reperfusion period. Furthermore, the recovery rate for spontaneous rhythm was significantly higher in animals given lidocaine. Praeger and co-workers [11] gave lidocaine intravenously just before aortic declamping to 100 patients having elective myocardial revascularization. They observed a significant reduction in the incidence of ventricular fibrillation after reperfusion and in the mean number of countershocks required to defibrillate the heart in those 100 patients compared with 94 patients not receiving lidocaine. In the present study, 18 of 23 patients receiving lidocaine had spontaneous rhythm recovery, and no patient had permanent heart block.

One advantage of the spontaneous resumption of ventricular rhythm without ventricular fibrillation is that the need for electrical countershocks is obviated in the majority of patients. Although a single countershock is a routine maneuver in many institutions, it has been suggested that multiple countershocks may be injurious to the heart. In our study, we observed nearly a fourfold increase in the number of countershocks required to defibrillate the hearts of patients not receiving lidocaine blood cardioplegia. Using isoenzyme measurements, technetium-labeled pyrophosphate imaging, and histopathological methods as indicators of damage, Dahl and associates [12] demonstrated substantial myocardial damage in patients receiving multiple electrical countershocks.

The second presumed drawback of local anesthetics is left ventricular function depression after reperfusion. Experimentally, Hearse and co-workers [13] demonstrated improved ventricular function in the isolated rat heart model when lidocaine was added to St. Thomas' cardioplegic solution. Clinically, we found that left ventricular function was minimally depressed at 30 minutes after reperfusion, although the effect was transient and appeared to be of no clinical importance. A similar transient reduction in left ventricular function was noted by Binnion and colleagues [14] in their canine model. Right ventricular function in the present study was not affected by the lidocaine infusion. Sunamori and co-workers [15] used continuous intravenous administration of lidocaine (1 mg/min) in patients undergoing elective myocardial revascularization. They saw no impairment in left ventricular function; rather, they documented improvement in stroke volume and cardiac index with a significant reduction in the CK-MB release in patients receiving lidocaine compared with controls. In our study, we also noted a trend toward less CK-MB release in patients receiving lidocaine blood cardioplegia, but the difference did not achieve significance, possibly because of the small sample size.

Lidocaine appears to be a safe and beneficial additive to blood cardioplegia for most patients. Contraindications are few. Hypersensitivity or allergic manifestations to local anesthetics are virtually limited to the ester-linked agents (eg, procaine), whereas the amide-linked local anesthetics such as lidocaine are singularly free from such deleterious effects [16]. Because lidocaine is almost exclusively metabolized in the liver, patients with advanced liver disease have greatly reduced lidocaine clearance. In this setting, we consider it safer to eliminate lidocaine from the cardioplegic solution.

This study suggests that blood cardioplegic solutions containing lidocaine result in prompt resumption of sinus rhythm after cross-clamp removal in the majority of patients. When this solution is used, ventricular fibrillation occurs uncommonly and electrical countershock is unnecessary in most patients. At the dose levels reported in this study, there appears to be no clinically significant or sustained left ventricular dysfunction. The improvement in CK-MB release, though not significant, suggests a trend toward less myocardial damage in those patients receiving lidocaine cardioplegic solutions. This preliminary report warrants further investigation of the use of lidocaine in cardioplegic solutions.

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Internal Mammary Artery Revascularization in the Patient on Long-Term Renal Dialysis

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Twenty-six patients on long-term renal dialysis underwent coronary artery bypass grafting. The patients were divided into two groups: group 1, (16 patients) saphenous vein bypass grafts, and group 2, (10 patients) internal mammary artery in combination with saphenous vein bypass grafts. Both groups were similar in terms of cardiac hemodynamics and previous number of myocardial infarctions, though more group 1 patients were in New York Heart Association class III or IV. Patients in group 1 received 2.9 bypass grafts per patient; patients in group 2 received 4.0 bypass grafts per patient (4 with bilateral mammary arteries). No wound healing problems occurred in either group. Blood replacement was similar for both groups (group 1, 5.5 units/patient; group 2, 5.3 units/patient). More platelets were given to group 1 patients (16.2 units/patient) than group 2 patients (3.1 units/patient). We conclude that use of the internal mammary artery in patients on long-term renal dialysis does not alter wound healing or increase blood loss in this subset of patients.

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Multiple reports [1–19] currently exist in the literature stating that coronary artery bypass grafting and valve replacement can be performed with a reasonable morbidity and mortality in patients on long-term renal dialysis. Patients to date have been revascularized only with saphenous vein bypass grafts, despite improved patency rate and long-term patient survival noted with internal mammary artery revascularization and possibly with the gastroepiploic artery [20–28]. The presumed reluctance for use of the internal mammary artery is poor wound healing in patients on renal dialysis and increased bleeding due to platelet dysfunction. Of the 31 patients on renal dialysis operated on at Loyola University Medical Center, 26 had coronary revascularization, and 10 of these patients were revascularized with at least one internal mammary artery. We compared perioperative bleeding, blood product replacement, and wound healing between patients receiving saphenous vein revascularization only and patients receiving internal mammary artery revascularization in combination with saphenous vein bypass grafts.

Material and Methods

Thirty-one patients on long-term renal dialysis had 32 cardiac procedures performed. Twenty-six procedures were coronary revascularization. Group 1 consisted of 16 patients receiving saphenous veins only as the method of revascularization. Group 2 consisted of 10 patients who received a combination of saphenous vein and internal mammary artery revascularization. Three patients in group 2 had bilateral internal mammary artery grafts as a method of revascularization, 1 patient had an aortic root enlargement and valve replacement with an internal mammary artery bypass graft, and 1 patient received bilateral internal mammary artery grafts in combination with a gastroepiploic artery and two saphenous veins (Table 1). The remaining patients received one internal mammary artery bypass graft in combination with saphenous veins. Group 1 had a mean age of 60.8 years and consisted of 9 men and 7 women. Group 2 had a mean age of 55 years and consisted of 7 men and 3 women. These patients were operated on over a time span of 10 years beginning in 1981.

All patients underwent dialysis the day before operation, and renal dialysis was reinstituted the day after operation. Blood replacement was done as necessary to maintain a hematocrit postoperatively of 0.24. Other blood products—platelets, fresh frozen plasma, and cryoprecipitate—were administered as deemed necessary by the operating surgeon. Surgical technique consisted of systemic cooling down to 28°C and use of blood cardioplegia with a hematocrit of 0.10. Wound closure for all patients was performed by the same method: sternal rewiring and a subcutaneous closure in several layers using a Dexon running suture for both the sternal wound and the legs.

Twelve patients in group 1 were classified as in New York Heart Association class III or IV, and only 4 patients in group 2 were in the same class. Mean pulmonary artery pressure for group 1 was 40/18 mm Hg, wedge pressure was 17 mm Hg, ejection fraction was 0.47 for the left ventricle, and cardiac index was 2.6 L/m^2. Mean pulmonary artery pressure for group 2 was 40/14 mm Hg, wedge pressure was 13.7 mm Hg, ejection fraction was 0.41 for

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Table 1. Method of Revascularization for Group 2

Patient	Method
AS	Bilateral IMAs, gastroepiploic, 2 veins
JW	LIMA, 1 vein, aortic root enlargement with AVR
DP	Sequential LIMA, RIMA, 2 veins
RW	Bilateral IMAs
ET	Bilateral IMAs, 4 veins
DG	LIMA, 4 veins
CC	LIMA, 2 veins
LS	LIMA, 4 veins
LC	LIMA, 3 veins
JM	LIMA, 2 veins

AVR = aortic valve replacement; IMA = internal mammary artery; LIMA and RIMA = left and right internal mammary artery.

the left ventricle, and cardiac index was 3.0 L/m^2. Though the groups are similar hemodynamically, more patients in group 1 had unstable angina accounting for more patients in New York Heart Association classes III and IV. Seven group 1 patients had had previous myocardial infarction, and 5 group 2 patients had also had myocardial infarctions. The mean number of bypass grafts performed was 2.9 for group 1 and 4.0 for group 2.

Results

All 26 patients survived the perioperative period and were discharged home. No wound healing problems occurred in either group. Two patients in group 1 were returned to the operating room for excessive bleeding, and no patient in group 2 was returned to the operating room for bleeding.

Blood replacement for group 1 consisted of an average of 5.5 units of packed red blood cells, 2.3 units of fresh frozen plasma, 16.2 units of platelets, and 2.1 units of cryoprecipitate for the intraoperative and perioperative period. Group 2 patients required 5.3 units of packed red blood cells, 3.1 units of platelets, and 1 unit of fresh frozen plasma for the same time period. Chest tube drainage was more in group 2, with 1,804 mL of drainage before the chest tubes were removed compared with 738 mL of fluid for group 1.

Sixteen complications occurred in group 1 compared with four complications in group 2. Complications in group 1 consisted of atrial arrhythmias (3 patients), pneumonia (2), bleeding (2), seizure (2), perioperative myocardial infarction (1), ventricular tachycardia (1), deep venous thrombosis (1), urinary tract infection (1), clotted fistula (1), septicemia (1), and foot drop (1). Complications in group 2 consisted of pericardial effusion, pneumonia, urinary tract infection, and leg wound hematoma (1 each). None of the complications was life-threatening. The postoperative stay in the hospital was similar at 18.1 days for group 1 and 16.3 days for group 2.

Follow-up data for group 1 at a mean of 16.5 months demonstrated that 7 patients had died, only 1 due to a known cardiac problem. Two patients died secondary to sepsis, and two died due to gastrointestinal bleeding. The other causes of death could not be documented. The other patients are alive and doing well. Follow-up data for group 2 patients at a mean of 8 months reveal that 1 patient died due to sepsis 3 months after operation and the other 9 patients are alive and have no cardiac-related symptoms. Eight of the group 2 patients are candidates for renal transplantation, and to date, 2 have successfully undergone transplantation.

Comment

Methods of revascularization have improved markedly during the past two decades. The best method of revascularization as defined by patency at a 10-year follow-up is use of the internal mammary artery [20–23]. Furthermore, there does not appear to be an increased morbidity or mortality with the use of the internal mammary artery [24, 25]. The one exception may be the older diabetic patient [24]. The issue of using the internal mammary artery for patients on long-term dialysis has not been addressed in the literature [1–19]. The presumed reluctance is twofold: because of impaired wound healing in the patient with renal failure and because of less effective clotting due to platelet dysfunction, which may cause the patient to bleed more from the chest wall.

Despite these potential problems and because many of our recent patients were candidates for renal transplantation, we decided to take an aggressive approach to revascularization by using one or more internal mammary arteries and the gastroepiploic artery when appropriate. Historical controls in our own institution would then allow a similar group of patients for comparison. Though this group of patients may seem small, the largest number of patients on long-term dialysis reported to date by a single institution is 39 [17].

The groups were similar in terms of cardiac hemodynamics and in terms of previous myocardial infarcts. Group 1 patients did have a higher percentage of patients in New York Heart Association class III or IV because more patients had unstable angina.

No wound healing problem existed in either group for the chest incision or the leg incision. It should be further noted that 5 group 2 patients were also insulin-dependent diabetics. Blood replacement for the two groups was similar for packed red blood cells; however, group 1 patients required nearly five times the number of platelets and twice as much fresh frozen plasma. Many more units of platelets and fresh frozen plasma were given in our early experience with patients with renal failure for anticipated bleeding than actual bleeding. Platelets are now given only if the patient shows signs of excessive bleeding in the postoperative period. Only 3 of the patients in group 2 subsequently needed platelet transfusions.

Though blood replacement was less, chest tube drainage was more excessive in group 2. The reason is not certain, but it has been our general experience that patients with internal mammary artery dissection have more serous drainage in the perioperative period. Though the postoperative hospitalization period was similar between the two groups, many more complications did occur in group 1. Again, the reasons are not fully explainable. It should be remembered that none of the problems were life-threatening. The only notable difference between the groups was that many more patients in group 1 had unstable angina, but this does not account for the increased morbidity in this group.

One disturbing issue is that 7 group 1 patients died, only one due to a cardiac problem. As noted in a previous publication, this does not greatly alter Comty's figure of 25% mortality per year for people with known coronary artery disease on long-term dialysis who have not received bypass grafts. Patients undergoing coronary artery bypass grafting who have had kidney transplants have a significantly improved prognosis [2, 6, 7]. Because 8 of the patients in group 2 are candidates for renal transplantation, we anticipate an improved long-term prognosis for this group.

Finally, the issue must be addressed when to use the internal mammary artery in this group of patients. It is the belief of the lead author (B.P.B.) that its use should be applied to all dialysis patients unless they are hemodynamically unstable or have received prior irradiation to the chest. The fact that no internal mammary arteries were used in group 1 reflects more the personal philosophies of the surgeons involved.

In conclusion, patients with renal failure should not be denied coronary revascularization with the internal mammary artery because of presumed problems with wound healing or bleeding. Our small group of patients on dialysis demonstrates no increased morbidity or mortality because the internal mammary artery was used.

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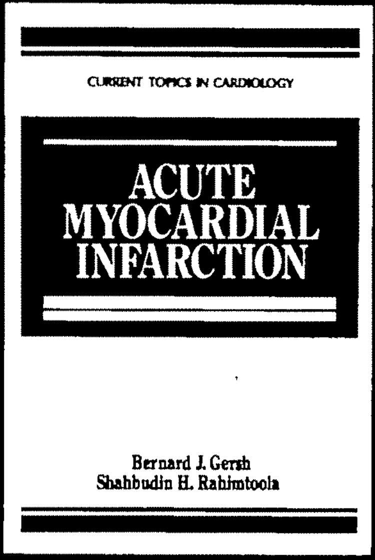

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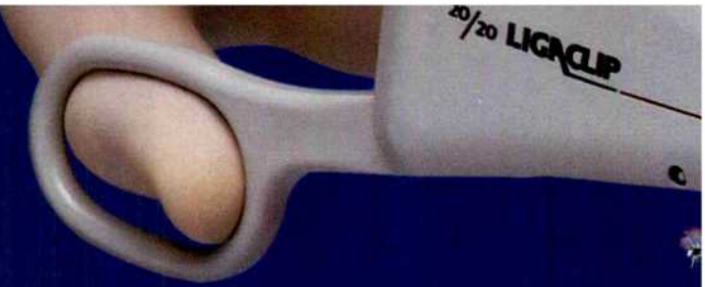

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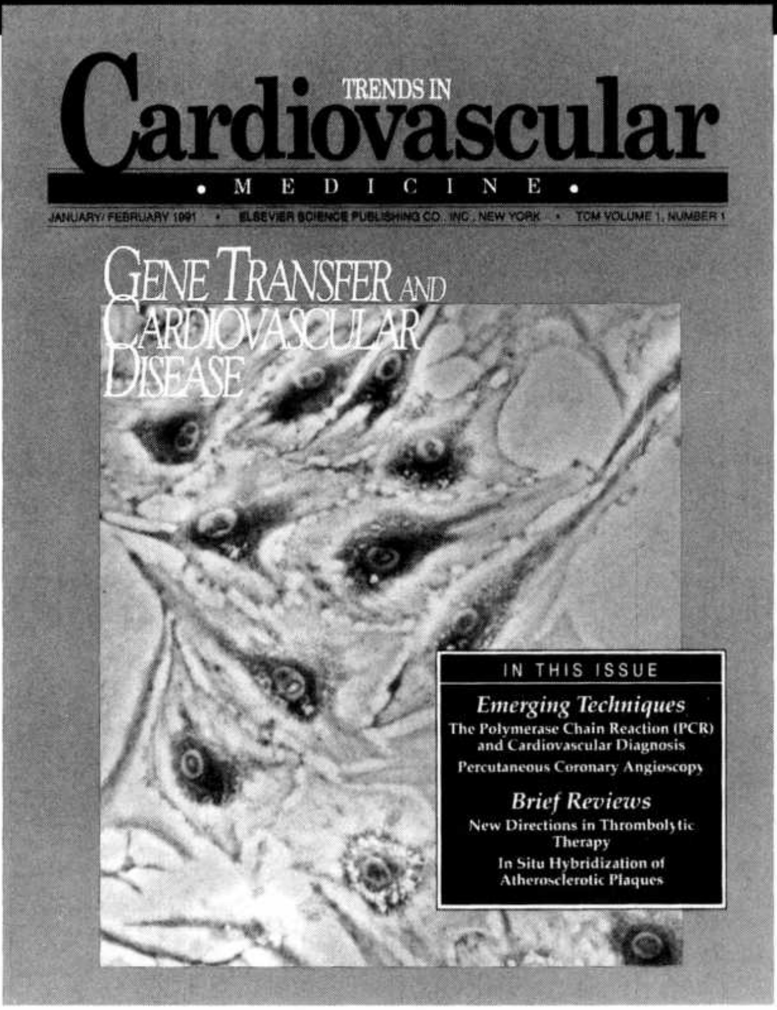

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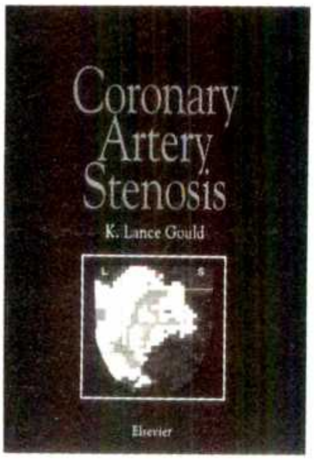

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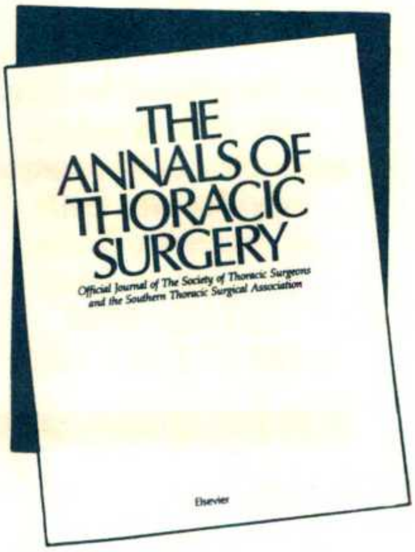
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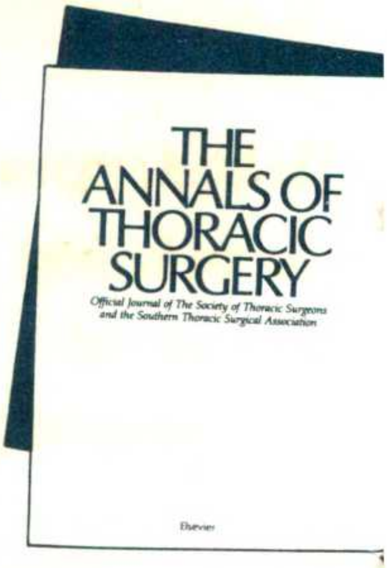
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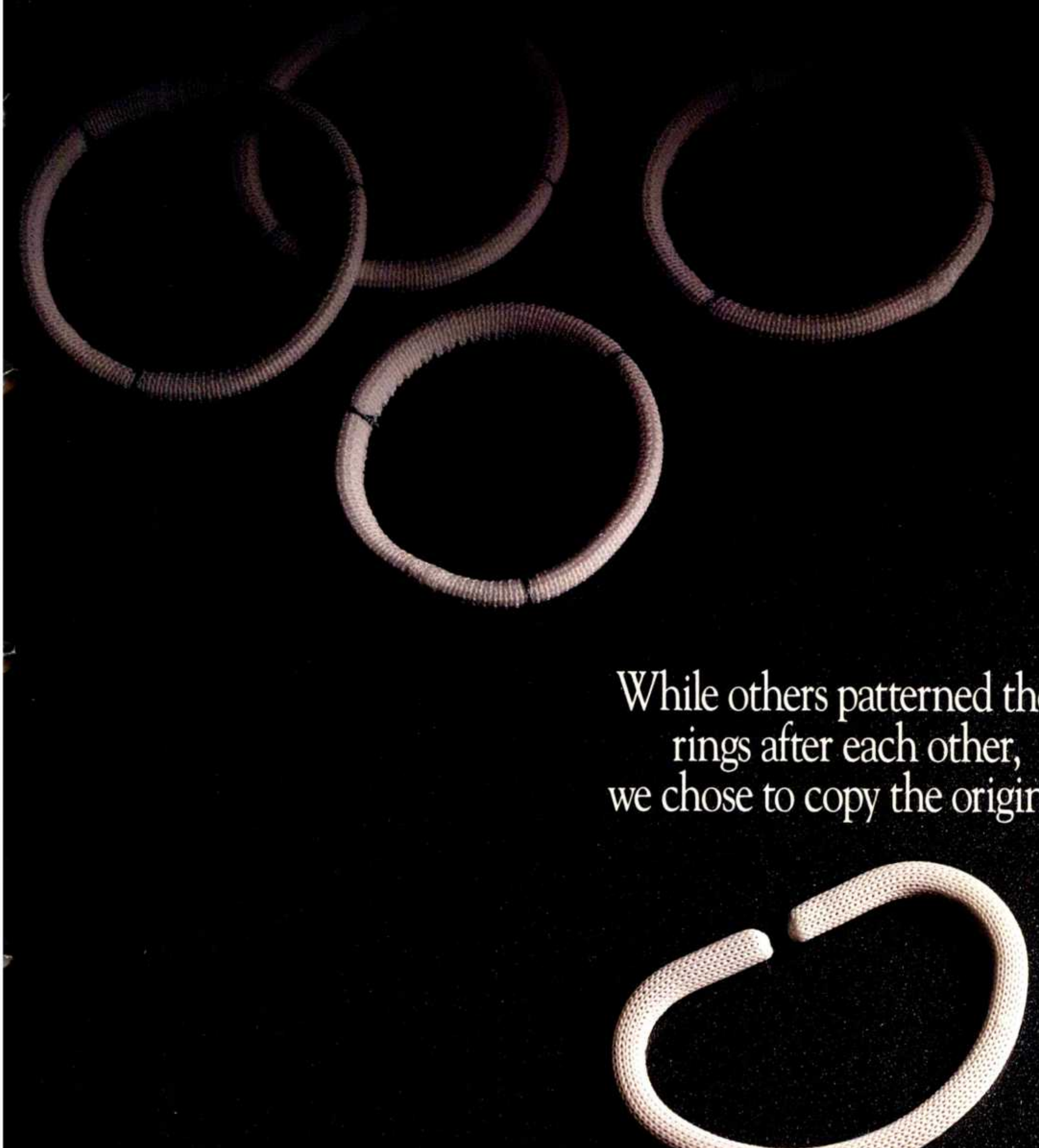
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Timing of Operation for Aortic Regurgitation: Relation to Postoperative Contractile State

Kazuhiro Taniguchi, MD, Susumu Nakano, MD, Hikaru Matsuda, MD, Yasuhisa Shimazaki, MD, Kei Sakai, MD, Tomohide Kawamoto, MD, Shigehiko Sakaki, MD, Junjiro Kobayashi, MD, Hideo Shintani, MD, Masataka Mitsuno, MD, and Yasunaru Kawashima, MD

First Department of Surgery, Osaka University Medical School, and Division of Cardiovascular Surgery, Sakurabashi-Watanabe Hospital, Osaka, Japan

With angiography and pressure measurement, we determined left ventricular volume, wall stress, and systolic performance in 30 patients with aortic regurgitation before and after successful aortic valve replacement. End-systolic wall stress was greatly elevated preoperatively and decreased to normal postoperatively. Systolic pump performance assessed as ejection phase indexes was severely depressed preoperatively and improved to normal or near-normal postoperatively in most patients. The ratio of end-systolic wall stress to end-systolic volume index (ESS/ESVI), an index of myocardial contractility, was greatly decreased before operation. Postoperatively, the ratio increased in all patients, becoming normal in 12 of the 13 patients who had a preoperative ESS/ESVI of 2.9 or greater. However, 15 of 17 patients in whom the ESS/ESVI ratio was less than 2.9 still had subnormal ratios, which indicates the presence of irreversible contractile dysfunction. Stepwise multivariate analysis showed that preoperative ESS/ESVI was the only independent discriminator of postoperative normalization of the contractile function as assessed by ESS/ESVI. After aortic valve replacement, myocardial contractile state does not return to normal in a considerable number of patients. It is important to offer aortic valve replacement for aortic regurgitation before the chance for a good functional result is lost. The ESS/ESVI ratio may be a useful index in determining the timing of operation in patients with aortic regurgitation.

(Ann Thorac Surg 1990;50:779–85)

In patients with chronic aortic regurgitation, the onset of irreversible left ventricular dysfunction is often insidious. Several guidelines have been presented for timing of aortic valve replacement in relation to the surgical outcome [1–6]. If the surgical correction is performed before left ventricular contractile dysfunction becomes irreversible, patients may be expected to have a good outcome in terms of postoperative left ventricular function, symptoms, and survival. Therefore, which indexes of left ventricular contractile function would be useful in predicting surgical outcome is a challenging problem.

Standard ejection phase indexes such as ejection fraction and mean velocity of circumferential fiber shortening are strongly influenced by preload and afterload, and reflect overall ventricular performance rather than myocardial contractile state. End-systolic volume index is independent of preload and has been shown to be a better predictor of surgical outcome in valvar regurgitation than ejection fraction [5]. In a previous study [7], end-systolic volume index separated patients who had a satisfactory surgical result in terms of preservation of postoperative left ventricular function from those with an unsatisfactory result.

Recently, end-systolic volume index corrected for end-systolic stress (the ratio of end-systolic stress to end-systolic volume index [ESS/ESVI]) has been postulated to be a useful indicator of contractile function in situations where loading conditions are altered [8–11], and proved to be a good predictor of surgical outcome in patients with mitral regurgitation [9]. In the present study, we evaluated left ventricular contractile state before and after aortic valve replacement in patients with aortic regurgitation by using the ESS/ESVI ratio as a measure of myocardial contractility and tested this ratio as a predictor of postoperative functional result. The purpose of this study was to clarify a level of left ventricular dysfunction that predicts impaired postoperative left ventricular contractile state, and to thereby determine a guideline for aortic valve replacement in patients with aortic regurgitation.

Material and Methods

Patients and Measurements

The study population consisted of 30 patients with isolated, chronic aortic regurgitation who underwent cardiac catheterization before and a mean of 26 months (range, 6 to 80 months) after aortic valve replacement. There were 26 men and 4 women, with a mean age of 43 ± 12 years at the time of operation. All patients underwent valve replacement with a Björk-Shiley tilting-disc prosthesis. Indications for postoperative cardiac catheterization were

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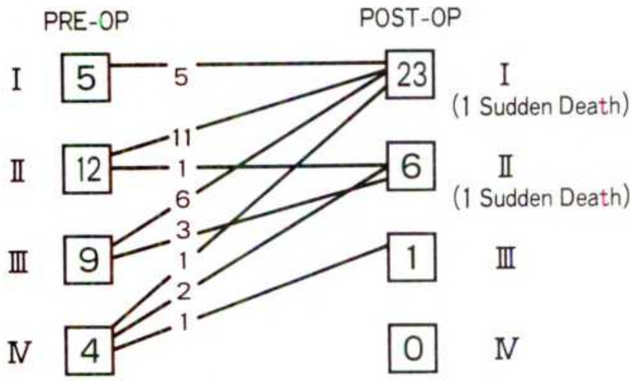

Fig 1. Preoperative and postoperative New York Heart Association functional classification of 30 patients with chronic aortic regurgitation.

nonselective, and patients who gave informed consent underwent the study. Patients with clinically significant aortic stenosis, other valvar lesions, or coronary artery disease were excluded. Patients were also excluded if high-quality left ventricular cineangiograms adequate for volume and stress analyses were not available. No patient had evidence of perioperative myocardial infarction or other serious complications such as the development of coronary artery disease during the period of observation.

The cause of aortic regurgitation was thought to be rheumatic disease in 19 patients, remote aortic valve endocarditis in 5, congenital bicuspid valve in 2, syphilis in 2, annuloaortic ectasia in 1, and Takayasu's aortitis in 1. Preoperatively, 5 patients were asymptomatic, 12 were minimally symptomatic (in functional class II of the New York Heart Association classification), and 13 were severely symptomatic (in class III or IV).

All patients underwent right and left heart catheterization and cineangiography. Our cardiac catheterization techniques have been published [7, 12] and are described only briefly herein. Pressures were measured with a micromanometer-tipped catheter (Millar Instruments, Inc, Houston, TX) or through a well flushed fluid-filled catheter connected to a Statham P23Db or P23ID strain gauge (Spectramed Inc, Oxnard, CA), and tracings were recorded at a paper speed of 100 mm/s with an 8-channel optical recording system. Aortic and left ventricular pressures were measured just before left ventriculography. Cardiac output was determined by the dye-dilution method. Aortic root angiography was carried out on all patients to evaluate the degree of aortic regurgitation.

Biplane left ventriculography was performed in the right anterior oblique (30 degree) and left anterior oblique (60 degree) projections for 24 patients, and single plane ventriculography was performed in the right anterior oblique (30 degree) projection for 6 patients. Cardiac volumes were obtained from the right anterior oblique ventriculograms. Volumes were calculated by the area–length method and were corrected by the regression equation derived by Wynne and associates [13]. Correction factors for x-ray magnification and pincushion distortion were obtained by use of a 1-cm^2 calibrated grid system. Extrasystolic and postextrasystolic beats were excluded, and the earliest satisfactory beat was selected for analysis.

End-systolic volume (ESV) was defined as the smallest ventricular volume, and end-diastolic volume (EDV) as the largest ventricular volume from serial frames. Ejection fraction was calculated as (EDV − ESV)/EDV. Wall thickness was measured at the mid anterior wall of the right anterior oblique ventriculogram at end-diastole. Left ventricular mass was determined at end-diastole by the method of Rackley and co-workers [14]. Volumes and mass were indexed for body surface area.

Mean velocity of circumferential fiber shortening was determined as $(Dd - Ds)/(Dd \times ET)$, where Dd and Ds = left ventricular minor-axis dimensions at end-diastole and end-systole, respectively, and ET = ejection time. Ejection time was obtained from the aortic pressure tracing recorded just before ventriculography. End-systolic circumferential wall stress was calculated from Mirsky's formula [15] as $(Pb/h)(1 - b^2/2a^2 - h/2b + h^2/8a^2)$, where p = left ventricular systolic pressure, h = end-systolic wall thickness, and a and b = the midwall semimajor and semiminor axes at end-systole, respectively. The result obtained by this formula was converted to dynes × $10^3/cm^2$ by multiplying by 1,332 dynes $\cdot$ cm^{-2} $\cdot$ mm Hg^{-1}. End-systolic wall thickness was calculated from end-systolic volume and left ventricular mass, which is assumed to be constant, according to the method of Hugenholtz and colleagues [16]. In the present study, left ventricular peak systolic pressure was substituted for end-systolic pressure. The ratio of end-systolic stress to end-systolic volume index was then calculated as a measure of myocardial contractility.

Postoperative left heart catheterization was performed with the transseptal technique. All prosthetic valves were considered to be functioning normally. Simultaneous pressure measurement for aortic and left ventricular pressures demonstrated peak systolic gradients across the prosthetic valve less than 10 mm Hg in 25 patients, 10 to 20 mm Hg in 3 patients, and greater than 20 mm Hg (22 and 30 mm Hg) in 2 patients. A paravalvular leak was ruled out in all patients by aortic root angiography.

Normal values for quantitative angiographic and left ventricular function data were obtained from 20 normal subjects who had undergone catheterization because of atypical chest pain syndromes. There were 15 men and 5 women, with a mean age of 43 ± 11 years.

Statistics

Data are expressed as mean ± standard deviation. Comparisons of preoperative and postoperative data were made using a paired Student's *t* test. Unmatched variables were compared by an unpaired *t* test. The normal range for a variable was defined as mean ± 2 standard deviations obtained in control subjects. Therefore, the lowest limit of normal for ESS/ESVI was calculated as 4.9. The significance of variables as independent predictors of postoperative contractility defined by ESS/ESVI was determined by stepwise multivariate discriminant analysis or stepwise multiple linear regression analysis.

Table 1. Preoperative and Postoperative Hemodynamic, Volume, and Mass Data

Group	HR (beats/min)	LVPSP (mm Hg)	LVEDP (mm Hg)	CI ($L \cdot min^{-1} \cdot m^{-2}$)	EDVI (mL/m^2)	ESVI (mL/m^2)	LVMI (g/m^2)
Aortic regurgitation (n = 30)							
Preoperative	80 ± 18	142 ± 20	15 ± 7	2.87 ± 0.94	248 ± 93	139 ± 76	285 ± 119
Postoperative	77 ± 17	141 ± 23	8 ± 3	3.55 ± 0.97	103 ± 31	45 ± 25	180 ± 70
p Value[a]	NS	NS	<0.01	<0.01	<0.01	<0.01	<0.01
Normal subjects (n = 20)							
Mean ± 1 SD	72 ± 14	126 ± 20	9 ± 3	3.85 ± 1.06	82 ± 13	28 ± 7	97 ± 11
p Value[b]	NS	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
p Value[c]	NS	<0.05	NS	NS	<0.01	<0.01	<0.01

[a] Comparison of preoperative versus postoperative values. [b] Compared with preoperative values in patients with aortic regurgitation. [c] Compared with postoperative values in patients with aortic regurgitation.

CI = cardiac index; EDVI = end-diastolic volume index; ESVI = end-systolic volume index; HR = heart rate; LVEDP = left ventricular end-diastolic pressure; LVMI = left ventricular mass index; LVPSP = left ventricular peak systolic pressure; NS = not significant; SD = standard deviation.

Results

Clinical Status

The New York Heart Association functional class had improved in most patients after aortic valve replacement (Fig 1). One patient was in class III and subsequently had progressive heart failure. Two patients died suddenly 28 and 57 months after postoperative catheterization.

Hemodynamics, Volume, and Mass

Left ventricular peak systolic pressure did not significantly change after operation (Table 1), whereas left ventricular end-diastolic pressure decreased and cardiac index increased significantly. Left ventricular end-diastolic volume index and end-systolic volume index decreased significantly after operation, as did left ventricular mass index.

Stress, Ejection Performance, and Contractile Function

End-systolic wall stress was substantially elevated preoperatively and decreased significantly after operation (Table 2, Fig 2). There was no significant difference between the patient group and control group.

Ejection fraction was markedly reduced preoperatively and increased significantly after operation. Likewise, the mean velocity of circumferential fiber shortening increased significantly to normal range in many patients.

The ESS/ESVI ratio was substantially reduced before operation (Fig 3). Postoperatively the ratio increased significantly in every patient. When a preoperative ratio cutoff value of 2.9 was selected, an excellent statistical separation was produced between two groups of patients with and without postoperative normalization of the ratio; the ESS/ESVI in 12 of the 13 patients who showed a preoperative ratio of 2.9 or greater returned to normal values (6.3 ± 1.3 versus 7.3 ± 1.2 for control subjects; not significant) postoperatively, whereas 15 of the 17 patients with a ratio less than 2.9 exhibited a persistently depressed ESS/ESVI ratio.

There was a strong correlation ($r = 0.79$, $p < 0.001$)

Table 2. Preoperative and Postoperative Wall Stress, Ejection Performance, and Contractile Function

Group	ESS ($kdynes/cm^2$)	EF	Mean VCF (circ/s)	ESS/ESVI
Aortic regurgitation (n = 30)				
Preoperative	303 ± 55	0.47 ± 0.11	0.79 ± 0.25	2.7 ± 1.1
Postoperative	193 ± 46	0.58 ± 0.11	1.16 ± 0.30	5.0 ± 1.9
p Value[a]	<0.01	<0.01	<0.01	<0.01
Normal subjects (n = 20)				
Mean ± 1 SD	199 ± 40	0.67 ± 0.06	1.15 ± 0.18	7.3 ± 1.2
p Value[b]	<0.01	<0.01	<0.01	<0.01
p Value[c]	NS	<0.01	NS	<0.01

[a] Comparison of preoperative versus postoperative values. [b] Compared with preoperative values in patients with aortic regurgitation. [c] Compared with postoperative values in patients with aortic regurgitation.

EF = ejection fraction; ESS = end-systolic wall stress; ESS/ESVI = ratio of end-systolic wall stress to end-systolic volume index; NS = not significant; SD = standard deviation; VCF = velocity of circumferential fiber shortening.

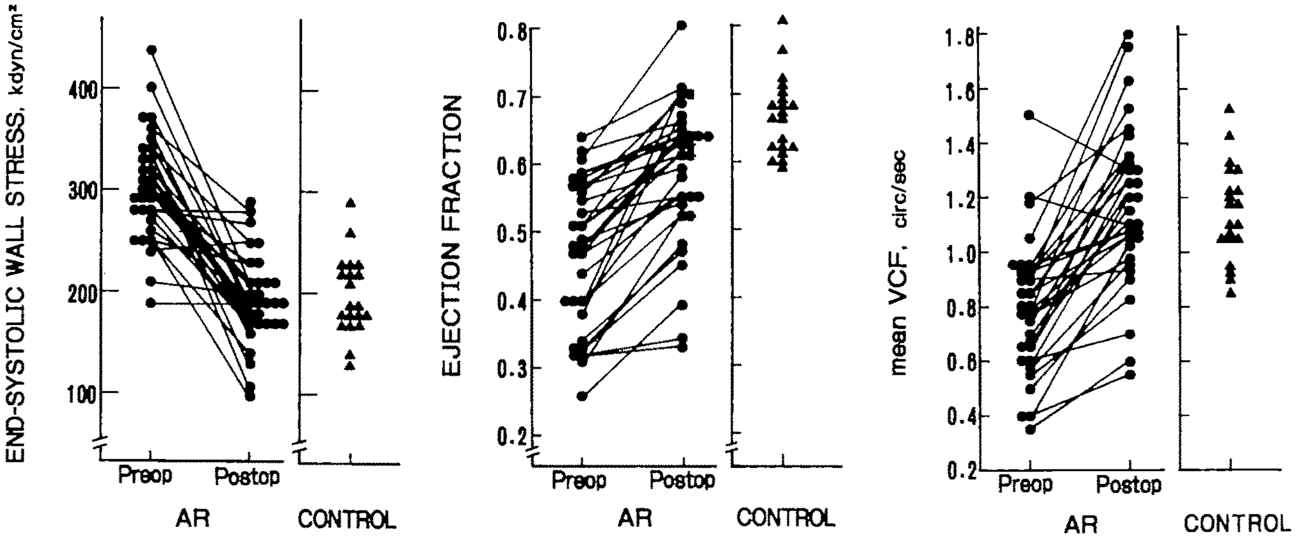

Fig 2. (Left) Left ventricular end-systolic wall stress decreased in all patients with aortic regurgitation (AR) postoperatively, becoming identical to the values in 20 normal subjects (CONTROL). (Center) Ejection fraction increased significantly, with 21 patients showing a normal ejection fraction (within 2 standard deviations of normal) after operation. (Right) Likewise, mean velocity of circumferential fiber shortening (mean VCF) *increased remarkably, returning to normal in all but 3 patients after operation.*

between preoperative and postoperative ESS/ESVI ratios. Although the preoperative ejection fraction was also related significantly to the postoperative ESS/ESVI, the corrrelation was less strong ($r = 0.68$, $p < 0.001$).

The postoperative values of ejection fraction and ESS/ESVI correlated well ($r = 0.70$, $p < 0.001$), but the relationship was nonlinear (Fig 4). Nine of 24 patients with a normal or near-normal ejection fraction (>0.50) had a depressed ratio (<4.9), whereas none of 15 with a normal ratio had a depressed ejection fraction.

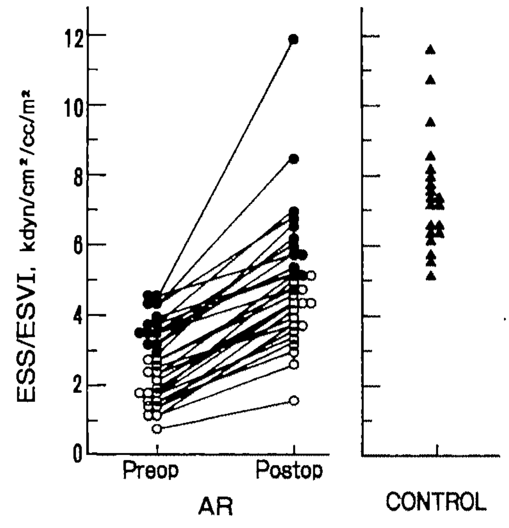

Fig 3. The ratio of end-systolic wall stress to end-systolic volume index (ESS/ESVI) was reduced in patients with aortic regurgitation (AR) before operation, and improved in every patient postoperatively. A preoperative ratio cutoff value of 2.9 permitted the best statistical separation of the two postoperative groups of patients with and without complete normalization of contractile function, as defined by a ESS/ESVI ratio of 4.9 or greater (control mean − 2 standard deviations). The postoperative ratio was normalized in all but 1 patient who showed a ratio greater than 2.9 preoperatively (closed circle)*, but remained abnormal in 15 of the 17 patients with a preoperative ratio less than 2.9* (open circle)*. The values for 20 normal subjects (CONTROL) are indicated in the extreme right column.*

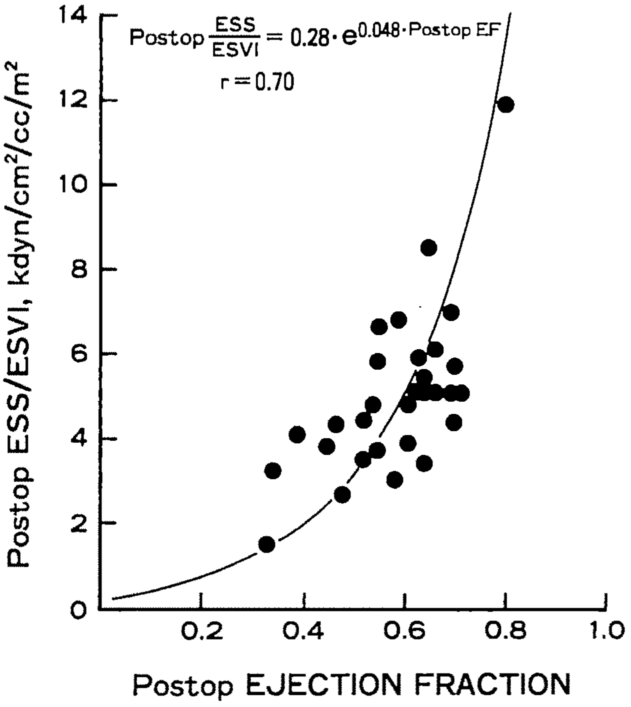

Fig 4. Relation between the postoperative ejection fraction and postoperative ratio of end-systolic wall stress to end-systolic volume index (ESS/ESVI). There was a wide range of ESS/ESVI values among patients with normal or near-normal ejection fraction.

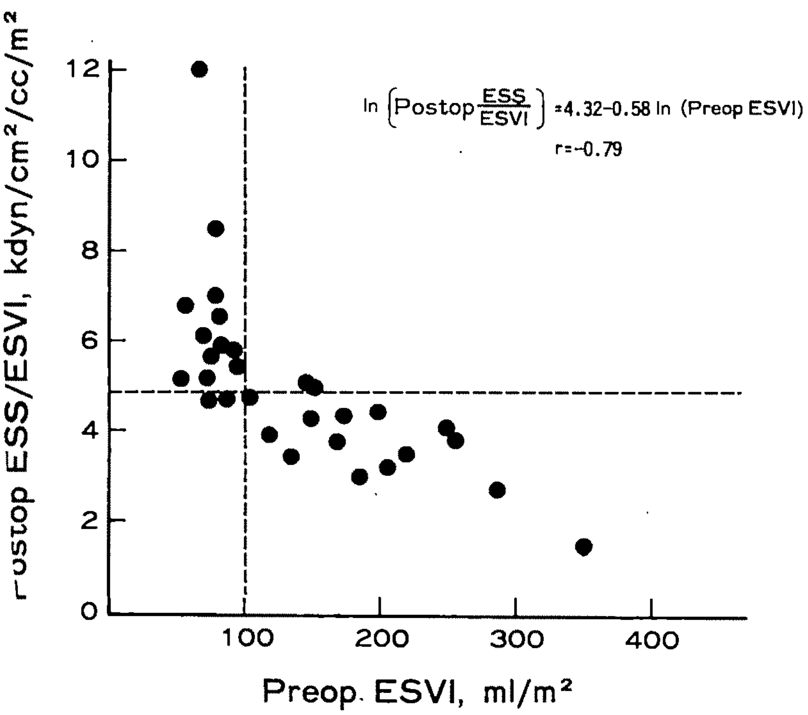

Fig 5. Relation between the preoperative end-systolic volume index (preop ESVI) *and postoperative ratio of end-systolic wall stress to end-systolic volume index* (postop ESS/ESVI). *Twelve of the 14 patients who had an ESVI less than 100 mL/m²* (vertical dashed line) *before operation achieved a postoperative normalization of the ratio* ($\geq$*4.9,* horizontal dashed line), *whereas all but 2 patients with an ESVI of 100 mL/m² or greater had a depressed ratio postoperatively. This analysis indicates that those patients with an ESVI of 100 mL/m² or greater are at high risk of persistent postoperative myocardial dysfunction.*

Predictive Value of Preoperative Variables for Postoperative Contractile State

Preoperative variables of age at operation, functional class, cardiothoracic ratio, left ventricular end-diastolic pressure, end-systolic and end-diastolic volume indexes, ejection phase indexes, degree of hypertrophy, end-systolic wall stress, ESS/ESVI ratio, size of Björk-Shiley prosthesis, and interval from operation to postoperative study were submitted to stepwise multivariate discriminant analysis. The preoperative ESS/ESVI was the only independent predictor (F value = 24.7, $p < 0.0001$) of postoperative normalization of the contractile state, as defined by a ESS/ESVI ratio of 4.9 or greater (control mean − 2 standard deviations). With a view for clinical use, we further examined the predictive value of other variables. When a stepwise multiple linear regression model (excluding ESS/ESVI) was employed, preoperative end-systolic volume index, rather than ejection fraction, was selected first by the model as an important predictor of postoperative contractile function (Fig 5).

As shown in Figure 6, there also was a significant correlation ($r = -0.93$, $p < 0.001$) between preoperative end-systolic volume index and preoperative ESS/ESVI ratio, which indicates that preoperative end-systolic volume index can be a practical alternative to measuring preoperative ESS/ESVI.

Comment

An important finding in this study was that preoperative ESS/ESVI ratio correlated well with postoperative contractile state in patients with aortic regurgitation. Our data support partly the finding of Carabello and associates [17] that ESS/ESVI was a useful predictor of postoperative prognosis or postoperative ejection fraction in patients with valvar regurgitation.

There have been a number of studies [18–26] that attempted to evaluate the effects of aortic valve replacement on left ventricular ejection performance and surgical outcome and thereby to determine the proper timing of operation for aortic regurgitation. Gault and co-workers [18] reported incomplete recovery of left ventricular function after successful valve replacement. On the other hand, many studies [12, 19–23] have demonstrated that impaired preoperative left ventricular ejection performance improves dramatically and even normalizes after operation. However, despite continued improvement in surgical results of valve replacement, there is a subgroup of patients who continue to exhibit left ventricular dysfunction and have an unfavorable outcome. Forman and colleagues [1] suggested that left ventricular ejection fraction was a useful predictor of surgical outcome. Graves and associates [2] reached a similar conclusion. Borow and co-workers [5] found end-systolic volume index to be a better predictor of outcome in valvar regurgitation than ejection fraction. On the other hand, Henry and associates [3] suggested that left ventricular end-systolic dimension more than 55 mm by M-mode echocardiography reliably identified a group with an unfavorable outcome. Fioretti and colleagues [27] found that a preoperative end-systolic dimension more than 55 mm did not preclude successful results, as judged by postoperative survival and improvement in left ventricular dimension. Thus, previous studies have had conflicting results. This may be attributed to the fact that ejection phase indexes are strongly influenced by preload and afterload conditions and therefore do not accurately reflect myocardial contractile state.

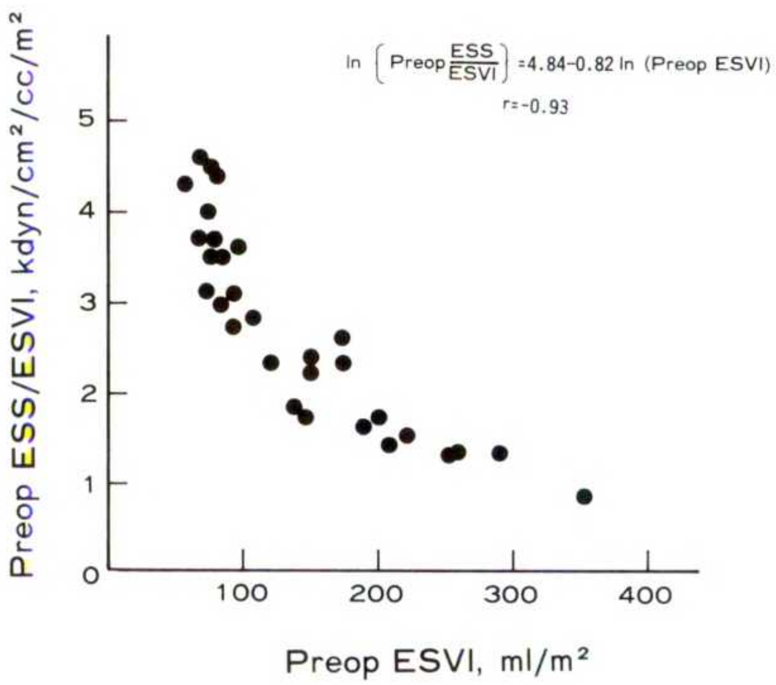

Fig 6. Relation between the preoperative end-systolic volume index (preop ESVI) *and preoperative ratio of end-systolic wall stress to end-systolic volume index* (preop ESS/ESVI). *As noted in previous studies [5, 7], ESVI may be a useful alternative to measuring the ESS/ESVI in patients with aortic regurgitation.*

In the present study, we evaluated left ventricular contractile state by the ESS/ESVI ratio. Carabello and co-workers [8] have suggested that ESS/ESVI is independent of preload and that, although partially afterload-dependent and inferior to the slope of end-systolic afterload–volume relationship (E_{max}) [11], it is clinically useful for estimating left ventricular contractile state. Ross [28] noted that the use of this ratio is desirable in evaluating the changes in the inotropic state for a chronically volume-overloaded ventricle before and after operation because substantial changes in ventricular size and wall thickness occur after surgical correction.

Our patients have experienced consistent but varying degrees of improvement in hemodynamic variables after operation. Afterload assessed as end-systolic wall stress fell and returned to normal ranges. Systolic pump function assessed as ejection phase indexes improved to normal or near-normal in many patients. These findings are consistent with results of previous studies.

Importantly, among our patients with postoperative normal or near-normal ejection performance, we identified patients who constituted two subgroups, one with preserved and the other with depressed contractile state (see Fig 4). All but 1 patient with a preoperative ESS/ESVI of 2.9 or greater had normal pump performance and preserved contractility after operation. In contrast, a considerable number of patients in whom preoperative ESS/ESVI was less than 2.9 had depressed contractile function after operation. These findings suggest that delaying operation in a patient with mild to moderate depression of myocardial contractility until the development of more severe contractile depression (that is, ESS/ESVI ratio $<$ 2.9) may place the patient at greater risk of irreversible postoperative myocardial dysfunction. Therefore, we believe that aortic valve replacement for aortic regurgitation should be performed before an ESS/ESVI becomes less than 2.9.

Our wall stress measurements have several limitations: (1) pressure and volume were not measured simultaneously, (2) pressures were measured with a fluid-filled catheter in many patients, and (3) peak systolic pressure was substituted for end-systolic pressure. However, as we have previously discussed in detail [12], it is doubtful that errors from these factors were so large that they would invalidate our study. The difference in peak systolic pressure with a well-flushed fluid-filled catheter and micromanometer-tipped catheter is small, and the time delay in transmission of pressure through fluid-filled catheters has been shown to be quite short (about 10 mm/s). Reichek and associates [29] showed a close correlation between peak systolic and end-systolic pressures ($r = 0.97$). End-systolic pressure has traditionally been approximated by aortic dicrotic pressure. In this study, when the ESS/ESVI ratio was calculated with aortic dicrotic pressure rather than peak systolic pressure, the results were qualitatively similar in terms of identifying patients with and without complete normalization of contractile state postoperatively. We have chosen peak systolic pressure as "end-systolic pressure" rather than aortic dicrotic pressure because precise aortic dicrotic pressure was difficult to define in some patients [29, 30].

The ESS/ESVI ratio is now known to be afterload-dependent and therefore less than ideal [11]. Recent reports have suggested that ESS/ESVI is considerably dependent on the ventricular size. However, because afterload returns toward normal and left ventricular size is reduced after operation, we believe that the postoperative ESS/ESVI can be a reliable indicator of postoperative contractile function, influencing postoperative survival. We found that preoperative ESS/ESVI and preoperative ejection fraction correlated significantly with the postoperative ESS/ESVI. These findings support strongly the concept that preoperative left ventricular systolic function is an important determinant of the surgical outcome.

The postoperative increase in ESS/ESVI ratio seen in our study may not necessarily mean improvement in contractility because of load- and size-dependence of the ratio. Unfortunately, we could not explicitly and definitively elucidate intrinsic changes in myocardial contractility in this study.

In conclusion, in patients with aortic regurgitation, preoperative ESS/ESVI was a useful predictor of outcome of valve replacement. An ESS/ESVI of less than 2.9 detected patients with an unfavorable result. Patients with a higher ESS/ESVI had a favorable result, suggesting normal postoperative contractile function. Aortic valve replacement should be recommended before the chance for postoperative recovery of contractility is lost.

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Thoracoscopic Treatment of Spontaneous Pneumothorax Using Carbon Dioxide Laser

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In an effort to improve the success rate of the previously described thoracoscopic electrocautery ablation technique of spontaneous pneumothorax, the carbon dioxide laser was evaluated in 12 patients. The recurrent (5 patients) or persistent (7 patients) spontaneous pneumothoraces were caused by rupture of (1) blebs in 6 patients, (2) intrapulmonary apical type II bullae in 3 patients, and (3) diffuse bullous emphysema, type III, in 3. The air leaks were successfully sealed in all but 1 patient with ruptured type II bulla. Surgical specimen from this single failure suggested that the entire inner lining of the bullae must be thermocoagulated. This technical modification led to successful outcome in 2 subsequent cases. With the use of carbon dioxide laser, it was possible to treat not only small blebs but all types of bullae causing spontaneous pneumothorax. Laser thoracoscopy is effective and safe in treating spontaneous pneumothorax.

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Earlier one of us [1] reported the use of an electrocautery through a thoracoscope in treating spontaneous pneumothorax. It was found that the electrocautery was effective in sealing ruptured blebs but failed to control air leaks from ruptured large bullae. In a search for a more effective means than the electrocautery, we evaluated the lasers. LoCicero and associates [2, 3] reported that the carbon dioxide (CO_2) laser is more effective in sealing air leaks from the cut surface of the lung than electrocautery. After extensive laboratory evaluation of the CO_2 laser [4, 5], we adopted it in place of the electrocautery. This report describes our preliminary clinical results. In order to describe our data, it is helpful to use our thoracoscopic classification of air-containing pseudocysts of the lung.

Air-containing pseudocysts of the lung vary in size and structure. Blebs are subpleural and small, less than 1 cm in diameter [6]. A ruptured bleb is not identifiable as a hole through a thoracoscope. Large pseudocysts (bullae) have three types. Type I is a thin-walled cyst with little communication with the bronchial system that is basically extrapulmonary in nature. Its lumen is singular and has no trabeculae. It is usually a few centimeters in diameter but sometimes attains a large size, 15 to 25 cm. At this size, such pseudocysts are readily visible on a plain chest x-ray film and cause compressive bullous emphysema. Surgical bullectomy for compressive bullous emphysema has been most successful for type I bullae. Type II pseudocyst is a medium-sized bulla and has thick, fibrous walls. It is located deep in the lung parenchyma (intrapulmonary bulla of Laurenzi and co-workers [7]). Only its superficial portion is visible through a thoracoscope. Its inside is separated by numerous septi [7]. Several type II bullae may be found together in one lobe. Patients with type II bullae remain asymptomatic and the bullae are not visible on plain chest films. When they rupture, causing spontaneous pneumothorax, however, surgical intervention is frequently required. A ruptured type II bulla is generally large enough to be seen through a thoracoscope. Type III is a large pseudocyst and is usually found in more than one lobe (diffuse bullous emphysema). They have multiple trabeculae and are the most common cause of diffuse bullous emphysema. Bronchial communications are abundant. When they rupture, surgical intervention is required, which carries a high morbidity and mortality rate [8, 9].

Material and Methods

Thoracoscopic CO_2 laser treatment was offered as an alternative to a conventional treatment such as chemical pleurodesis or thoracotomy and lung resection. A possibility of failure and the necessity for subsequent thoracotomy was explained to the patient. Only when the patient requested the laser thoracoscopy was this procedure performed.

The procedure was performed at two different hospitals, using different equipment. At hospital A, LaserSonics laser equipment (Cooper LaserSonics, Inc, model #SL780) was used with an operating laser laparoscope (model ML 107226A; Gynescope Corp, Willoughby, OH) and a video camera and recorder (Striker 570 CCD Plus; Striker Surgical, Kalamazoo, MI). This Striker video camera has no split-beam prism. At hospital B, an NIIC CO_2 laser system (model IR104; NIIC, Inc, Redwood City, CA) was used with an Olympus CO_2 laser laparoscope (model A5308L; Olympus Corporation, Lake Success, NY) with an Olympus Medical TV system (model #OTV-S2) and a video camera (model AR-TF2 with viewfinder). This video camera has a split-beam prism, thus the surgeon can see through the camera while the operative field is displayed on a monitor screen. Conventional laparoscopy accessories with electrocautery

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Table 1. Patient Data

Patient No.	Type of Pseudocyst	Indication	Age (y)	Sex	No. of Rx	Hospital
1	Bleb	Persistent	25	F	1	A
2	III	Recurrent	61	M	1	A
3	Bleb	Persistent	17	M	1	A
4	Bleb	Recurrent	20	M	3[a]	A
5	III	Persistent	64	M	3[a]	A
6	Bleb	Persistent	18	M	1	A
7	Bleb	Persistent	22	M	1	A
8	Bleb	Recurrent	28	M	1	B
9	II	Recurrent	24	M	3[b]	A
10	II	Persistent	19	M	1	A
11	III	Persistent	60	F	1	B
12	II	Recurrent	28	F	1	B

[a] Repeated thoracoscopies required due to inadequate visualization on a monitor. [b] Failure, requiring thoracotomy.

Rx = recurrences.

attachments, such as grasping forceps, suction probe, and scissors, were used during thoracoscopy. The CO_2 laser was used in a continuous wave and defocused mode. The calculated power density with the power setting of 20 W was approximately 250 W/cm^2 when the tip of the laserscope was held 3 cm from the target. For blebs, only a low power level (less than 15 W) was required. For type II or III pseudocysts, higher power levels (15 to 30 W) were used.

The patient was anesthetized, intubated with a split-lumen endotracheal tube, and placed in a lateral position. The ipsilateral lung was collapsed to obtain the maximal visualization of the lungs and to keep distance between the lung and the tip of the laserscope. Two small stab wounds were made by a trocar for the passage of the rigid scope and an operating probe in the fifth intercostal space at anterior and posterior axillary lines. Pleural adhesions were completely freed using endoscopic scissors or a suction probe with electrocautery. The CO_2 laser was fired at ruptured blebs or bullae to seal air leaks. All other intact blebs or bullae were also ablated by the laser. On completion of the intrathoracic procedure, a chest tube was placed through one of the stab wounds. The other holes were closed with stitches. The chest tube was connected to continuous suction until air bubbling ceased.

Twelve patients with spontaneous pneumothorax (Table 1) were treated with this technique. The cause of spontaneous pneumothoraces was ruptured blebs in 6 patients, ruptured type II bullae in 3, and ruptured type III bullae in 3. The indications for laser thoracoscopy were persistent pneumothorax for more than 5 days in 7 patients and recurrence within 6 months in 5. The average ages were 21.7 years for patients with blebs, 23.3 years for those with type II bullae, and 58.7 years for those with type III bullae (bullous emphysema).

Results

There were no deaths and no wound infection. In 5 of 6 patients with ruptured blebs, the air leaks were sealed without difficulty with the CO_2 laser. These patients were discharged home within 3 days. Two failures in 1 patient at hospital A were found to be caused by inadequate visualization through the video monitor. The third thoracoscopy under direct visualization without a video camera identified the missed air leak, which was readily sealed with the laser.

Spontaneous pneumothorax due to ruptured type II bulla developed in 3 young patients. In the first patient, pneumothorax recurred every time the air leaks were apparently sealed. After the failure of three laser thoracoscopies, thoracotomy and partial lung resection was strongly recommended against the patient's request for the fourth thoracoscopy. The surgical specimen revealed that only the extrapulmonary portion of the type II bulla was sealed and the entire inner surface of the bulla remained intact, including bronchial communications. This was the only patient who required thoracotomy in this series. Based on this finding, the technique was modified so that the entire inner surface of the bulla was exposed to the CO_2 laser. Two subsequent patients treated with this modified technique showed no air leaks on the next day after the laser treatment.

One of three patients with ruptured type III bulla also required three thoracoscopies due to inadequate visualization by the video monitor; at the third procedure the air leaks were successfully sealed. Two other patients underwent laser thoracoscopy, one with a direct vision thoracoscopy and the other with a video monitor, without difficulty. They were discharged home within 3 days. A referring physician of patient 11, who had severe emphysema, elected not to place a chest tube because this might lead to thoracotomy, which she would not tolerate. She had fixed right pneumothorax for 3 weeks before the thoracoscopic CO_2 laser sealing of the ruptured type III bulla (Figs 1, 2). No air leaks were noted the next day and she was discharged home 2 days after the treatment. The postoperative chest films revealed fully expanded emphysematous lungs (Fig 3).

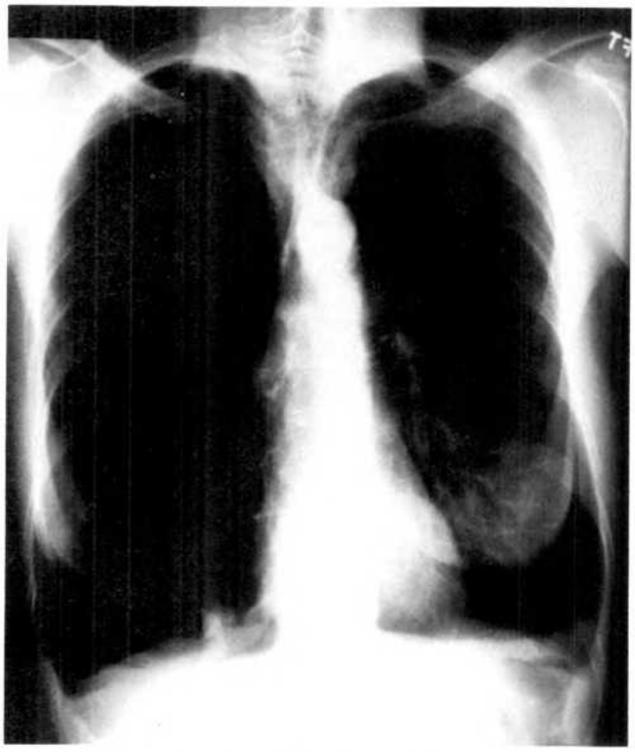

Fig 1. Preoperative chest x-ray film of patient 11 shows a fixed pneumothorax on the right side and underlying bullous emphysema.

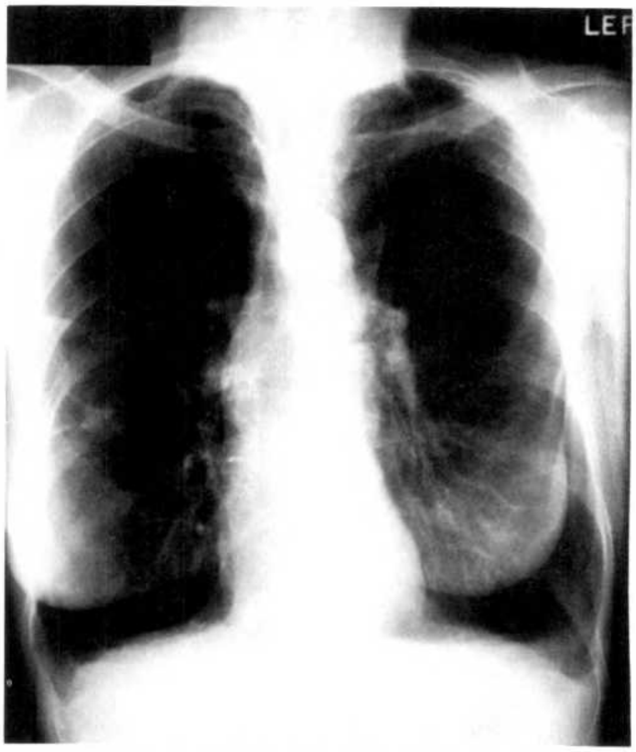

Fig 3. Postoperative chest x-ray film of patient 11 shows complete resolution of pneumothorax and the bullous changes in the bilateral upper lung fields.

Comment

Laser thoracoscopy was presented to the patient as an alternative to thoracotomy or pleurodesis. The patient was informed of our limited experience with this new technique, the up-to-date results, and possible complications, including the need for repeated laser treatment or subsequent thoracotomy and lung resection. The acceptance of the laser thoracoscopy by the patients was excellent and all of our patients had chosen it over other modalities even after it failed once or twice. The most common cause of failure was inadequate visualization provided by the video camera without a split-beam prism. All pleural adhesions must be freed to inspect the entire surface of the lung. Frequently, an air leak is hidden behind the fibrous adhesions. We prefer direct visualization over the video monitor as it provides a better and more clear picture.

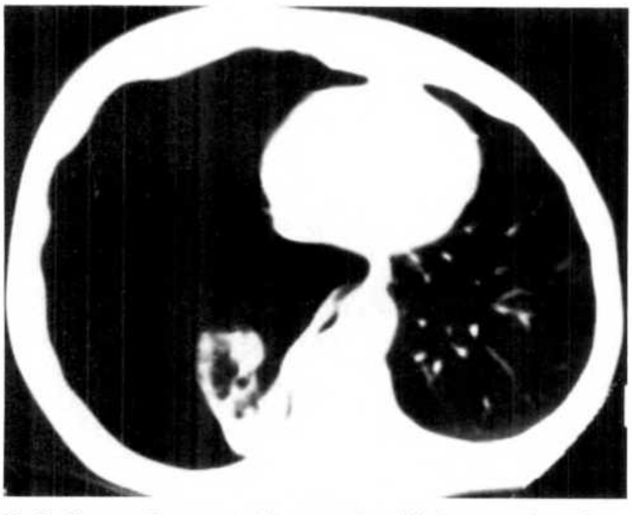

Fig 2. Preoperative computed tomography of the lungs confirms the findings in Figure 1 and the shift of the mediastinum to the opposite side.

In general, a ruptured bleb is not identifiable as a hole but is usually covered with fibrin deposits [1]. In contrast, a ruptured type II bulla can be identified as a 2- to 3-mm-diameter hole through a thoracoscope. With our previous [1] and current series combined, 4 of 19 young patients had this type of bulla (21%). The difficulty in treating type II bullae appears to have been solved, at least in part, by the application of the CO_2 laser beams to the inner surface, thus ablating the bullae from inside. However, if repeated laser thoracoscopy fails, thoracotomy and partial lung resection should be recommended without delay, as lung resection in this group of patients is well tolerated and effective. However, this was not the case with ruptured type III bullae. We observed that the CO_2 laser welds the trabeculae together and brings the surrounding lung tissues in, thus sealing the air leaks. It is our impression that thoracoscopic CO_2 laser sealing is more effective than suturing or staple closure of ruptured type III bullae. In our experience, more air leaks resulted from needle or staple holes placed through emphysematous lungs. Recently Bojar and Kittle [10] reported a low operative mortality rate (6%) after lung resection or suture closure of ruptured bullous emphysema in 16 elderly patients. However, this is not a universal experience and the morbidity and mortality rate in this group of patients still remains high.

Although electrocautery was as effective as CO_2 laser in ablating small blebs, it has no place in treating emphysematous bullae (type II or III). Because the presence of type II bullae is unpredictable and not uncommon, whenever the thoracoscopic approach is contemplated even in young patients, the CO_2 laser equipment should be available. Chemical pleurodesis is commonly used in treating spontaneous pneumothorax. It is our experience, however, that pleurodesis may not prevent recurrence of pneumothorax. Our patient 12, for instance, had tetracycline pleurodesis after the second episode of spontaneous pneumothorax. However, it recurred three more times before laser treatment.

In conclusion, our new technique of laser thoracoscopy, in which the CO_2 laser beams are guided through a rigid thoracoscope into the pleural space, was found to be a safe and effective treatment for all types of air-containing pseudocysts of the lungs causing spontaneous pneumothorax.

Dr Wakabayashi would like to express his deepest appreciation to Dr Paul Fuller at the Kaiser Permanente Anaheim Hospital for his expert advice.

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INVITED COMMENTARY

Traditionally, first-line treatment of a clinically significant pneumothorax (>20%) is reexpansion of the lung by either aspiration or tube thoracostomy [1]. When treated this way, a large portion of patients may even be managed with minimal hospital stays [2]. Successful reexpansion results in cure without recurrence in 80% of cases.

In the 20% of patients in whom a recurrence develops, there is agreement among most authors that operative intervention is appropriate [3–5]. Presently, the gold standard for operative therapy is a two-part procedure: bleb resection and pleurodesis. The latter may be accomplished in a variety of ways from talc poudrage to pleurectomy. The rationale for this approach is to eliminate the cause of the recurrent pneumothoraces, namely the bleb-containing area of the lung, and to prevent future lung collapse by a thorough pleurodesis.

Wakabayashi and colleagues propose to change the management of simple first-time pneumothorax in two important ways. First, they propose operative intervention in all cases. Any proposed procedure must show clear advantage over the already achieved cure rate of 80% with minimal morbidity. Presently, this series has an initial failure rate of 25%. Second, they propose bleb ablation as the sole method of surgical treatment. One might predict that if other blebs rupture in the future, recurrent pneumothorax may not be prevented. One year ago, Torre and Belloni [6] proposed similar early intervention for first-time pneumothorax using thoracotomy. They performed not only bleb ablation but also pleurodesis with the neodymium:yttrium-aluminum garnet laser in 14 patients without a recurrence.

These novel approaches are in keeping with the movement toward minimally invasive surgery. As lasers and other tools develop along with the instrumentation to deliver them into the chest, more innovative methods will surface. To become acceptable techniques, such procedures must undergo larger trials and stand the test of time to demonstrate their worth.

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REVIEW OF RECENT BOOKS

Interventional and Surgical Cardiovascular Pathology: Clinical Correlations and Basic Principles
By Fredrick J. Schoen
Philadelphia, W.B. Saunders
432 pp, illustrated, $75.00

Reviewed by Thomas A. Orszulak, MD

The author of this volume defined admirable goals in the introduction, striving for detailed coverage of clinical pathological correlations of cardiovascular disease and the pathology of physiological sequelae of these diseases. The chapters start with general pathological considerations of therapeutics, injury, and healing and address separately ischemic heart disease, valvar heart disease, myocardial, pericardial, and endocardial disease, mechanical assists and replacements, rhythm and conduction disturbances, and vascular and congenital heart disease. The appendix is specifically directed toward communication between pathologists and clinical services and research, with detailed description and an approach to postmortem examinations and specimens. The foremost goal of this treatise is to provide cardiovascular clinicians with the pathological data that will facilitate informed choices of therapeutic options for interventional procedures (by operation or by catheter) and permit optimal short- and long-term patient management and recognition of complications.

This goal is adequately met with detailed pathological information of disease states and results of therapeutic intervention. The most intriguing and informative chapters, in my estimation, are on ischemic heart disease and vascular disease. This may represent interest due to a practice pattern rather than deficiencies in the text. The text is by one author except the chapter on congenital heart disease. This provides a consistency in thought and organization throughout the text. The guest chapter on congenital heart disease follows this format and is exceptional for congenital pathological detail.

One aspect of the text that is disappointing is the interventional or surgical recommendations for the disease states discussed, primarily valvar heart disease. This information is derived from literature references and should be read with some healthy skepticism. These sections should have been prepared by an appropriate surgeon involved in those procedures (which would have substantially lengthened the text) or even deleted.

I think the text is good and would recommend its absorption by students in the fields of cardiovascular disease; I also recommend it to teachers in the field for its current information on correlation of pathology with interventional therapeutics.

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Prolonged Extracorporeal Life Support of Pediatric and Adolescent Cardiac Transplant Patients

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Douglas M. Behrendt, MD, Edward L. Bove, MD, G. Michael Deeb, MD,
Dennis Crowley, MD, Kathleen P. Heidelberger, MD, and Robert H. Bartlett, MD

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Options for mechanical support of pediatric patients with severe heart failure who are awaiting transplantation or have undergone transplantation are limited. This report examines 3 patients placed on extracorporeal life support (ECLS) while awaiting transplantation and 3 patients who underwent transplantation and suffered subsequent heart failure due to rejection or postoperative myocardial dysfunction. The overall survival rate was 2 of 6. The 2 surviving patients had a failing transplanted heart. There were no survivors among the patients placed on ECLS as a bridge to transplantation. In each case a contraindication to transplantation developed before a donor heart could be obtained. The mean time of ECLS support was 147.5 hours (range, 70 to 370 hours). The ECLS circuit did not affect cyclosporin levels or antirejection therapy. Extracorporeal life support can be used to support pediatric cardiac transplant patients with biventricular failure due to acute rejection or postoperative dysfunction. Although the results have been discouraging, ECLS may still have a role as a bridge to transplantation. However, complications can develop during ECLS that may preclude transplantation.

(Ann Thorac Surg 1990;50:791–5)

Cardiac transplantation in pediatric patients with heart failure recalcitrant to medical or surgical therapy has become increasingly common [1]. Deterioration of patients with severe heart failure is often rapid, with death or irreversible organ failure occurring before procurement of a donor organ. This problem has prompted interest in developing mechanical circulatory support devices to maintain patients until a suitable donor heart becomes available. Several mechanical support devices have been developed, including univentricular and biventricular assist devices, the total artificial heart, and the intraaortic balloon pump [2–4]. All have been used successfully in maintaining adult patients with life-threatening heart failure who are awaiting transplantation. A biventricular support device has also been used to support an adult patient with a failing transplanted heart [5].

Options for mechanical support of pediatric patients awaiting heart transplantation or with a failing transplanted heart are limited, however, owing to the size of the patients and the need for biventricular support. Intraaortic balloon pumps have been used in infants as small as 2 kg, but only partial circulatory support is offered by this device [6]. To date, ventricular assist devices and the total artificial heart are not available in appropriate sizes for pediatric patients. Venoarterial cardiopulmonary bypass via external catheters has been used to support neonatal and pediatric patients with cardiac and respiratory failure due to a variety of causes [7–11]. This procedure has been called extracorporeal membrane oxygenation; extracorporeal life support (ECLS) is a better term which will be used in this report. This is a report of our experience with ECLS in pediatric patients with heart failure who were awaiting transplantation or needed support of a failing transplanted heart.

Material and Methods

Patient Population

The charts were reviewed of 3 patients with a failing transplanted heart who required ECLS for circulatory support and 3 patients with heart failure who were placed on ECLS while waiting for a donor heart. All patients were on the Pediatric Cardiac Surgery service at C.S. Mott Children's Hospital. There were 4 boys and 2 girls, aged 7 months to 18 years. Weights ranged from 4 to 70 kg. Diagnoses are listed in Table 1. In each case venoarterial ECLS was initiated for cardiogenic shock that was refractory to optimum medical management.

The 3 patients placed on ECLS before anticipated transplantation had no contraindications to transplantation before the initiation of circulatory support. The patients who had received a cardiac transplant were on an immunosuppression regimen consisting of steroids, cyclosporin, and azathioprine. Two transplant recipients were supported for heart failure during a rejection episode, and right ventricular failure developed in 1 patient shortly after transplantation. In both cases of rejection the diagnosis was confirmed by biopsy demonstrating severe rejection. Treatment of rejection consisted of three meth-

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Table 1. Summary of Patient Data

Patient	Age	Sex	Weight (kg)	Diagnosis	Time on ECLS (h)	Hemofilter	Complications	Outcome
LQ	7 mo	F	4	RV failure of transplanted heart	64	Y	Bleeding	Survived
JG	3 y	F	14	Tx rejection	73	Y	None	Survived
MC	17 y	M	70	Tx rejection	112	Y	Neurological injury	Died
CG	15 y	M	38	Tx bridge (cardiomyopathy)	70	N	Bleeding, pulmonary edema	Died
RS	6 y	M	22	Tx bridge (double-outlet right ventricle; after Fontan)	196	N	Hemothorax	Died
BF	18 y	M	44	Tx bridge (situs inversus, single ventricle)	370	Y	Bleeding, sacral decubitus ulcer	Died

ECLS = extracorporeal life support; RV = right ventricular; Tx = transplantation.

ylprednisolone pulses (15 $mg \cdot kg^{-1} \cdot day^{-1}$) followed by antithymocyte globulin (15 $mg \cdot kg^{-1} \cdot day^{-1}$) in 1 patient and three methylprednisolone pulses followed by OKT3 in the other patient. The adequacy of OKT3 therapy was followed by flow cytometry. Lack of T cells indicated a therapeutic response to OKT3.

Extracorporeal Life Support Technique

The ECLS circuits used in this report were identical to those described in prior reports [7]. The circuit consisted of a membrane lung (SciMed Life Systems, Inc), a servo-regulated roller pump, and a heat exchanger. The circuit was primed with blood. Cannulation was performed under local anesthesia and intravenous sedation. The patient was paralyzed with succinylcholine and systemically anticoagulated with heparin (100 to 200 units/kg). The right internal jugular vein or femoral vein was cannulated for venous drainage. Oxygenated blood was reinfused through the axillary, carotid, or femoral arteries. Anticoagulation was maintained by constant heparin infusion to keep the activated clotting time between 200 and 240 seconds (between 180 and 200 seconds since 1988). Platelet counts were maintained at greater than 50 $\times 10^9$/L (50,000/μL) (greater than 100 $\times 10^9$/L since 1988). Blood was transfused as needed to keep the hematocrit above 0.40. Bypass flow was initiated at 75 to 100 $mL \cdot kg^{-1} \cdot min^{-1}$. Inotropes were weaned as tolerated. During ECLS the ventilator settings were placed at an inspired oxygen fraction of 0.30 to 0.40, peak inspiratory pressure of less than 30 cm H_2O, and a rate of 10 cycles/minute. Antibiotics and parenteral nutrition were administered throughout the ECLS course. Diuretics were given as needed to maintain urine output. When necessary, hemofiltration was instituted by placing a filter in the ECLS circuit.

Recovery of cardiac function was followed up by serial echocardiograms. Brief trial periods off ECLS and trends in the mixed venous oxygen saturation were used to determine when weaning of circulatory support could be initiated. Flows were gradually decreased to less than 50 $mL \cdot kg^{-1} \cdot min^{-1}$. Ventilatory support and inotropes were added as needed. When a longer trial off ECLS indicated that mechanical support was no longer necessary the cannulas were removed.

Results

Overall, 2 of 6 patients survived. Two of the 3 patients with a failing transplanted heart improved and were weaned off ECLS. There were no survivors among the patients placed on ECLS as a bridge to transplantation. In one survivor with pulmonary hypertension right-sided heart failure developed 30 hours after transplantation. She was supported for 64 hours on ECLS. She is alive with normal cardiac function 60 months later. The other survivor was on ECLS for 72 hours while being treated for a severe rejection episode. A photomicrograph of the cardiac biopsy specimen is shown in Figure 1. She subsequently survived for 8 months before dying suddenly of marked, diffuse coronary artery narrowing (Fig 2). One adult-sized patient with rejection initially had an intraaortic balloon pump placed but was converted to ECLS when shock persisted despite support with the balloon pump. He was maintained on ECLS for 112 hours while being treated for rejection. Cardiac function did improve, but he was electively removed from ECLS after sustaining a severe, irreversible neurological injury.

There were no survivors among the 3 patients placed on ECLS as a bridge to transplantation. The mean time of ECLS support for these patients was 195.3 hours, with a range of 79 to 370 hours. In each case ECLS was electively discontinued after contraindications to transplantation developed before a donor heart could be obtained. In one 15-year-old patient with familial cardiomyopathy, severe

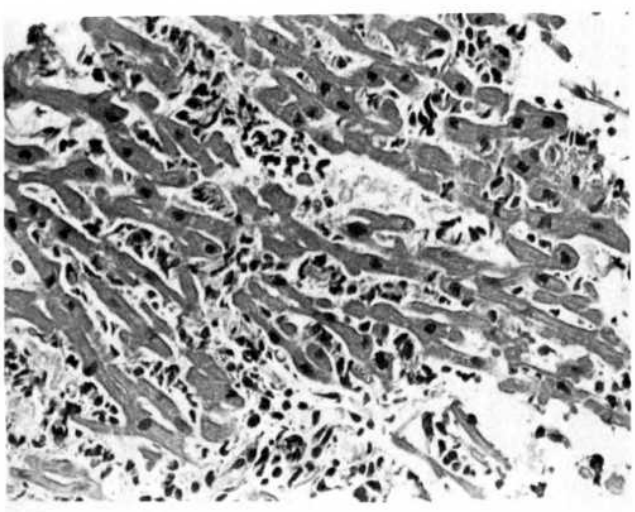

Fig 1. Biopsy specimen with severe rejection showing diffuse foci of infiltration by polymorphonuclear leukocytes and lymphocytes. Necrotic myofibers "dead end" in collections of inflammatory cells. (Hematoxylin and eosin, ×330 before 29% reduction.)

pulmonary edema developed after complete left ventricular failure developed during ECLS. Transatrial blade and balloon septostomy was performed 36 hours later in an effort to decompress the left atrium. This procedure failed to improve the pulmonary edema. Extracorporeal life support was discontinued when transplantation was no longer possible.

The second patient, an 18-year-old with situs inversus, levocardia, single ventricle, and a common atrioventricular valve was taken to the operating room for transplantation. However, the donor heart was found to be unsatisfactory. During anesthesia the patient's heart failure worsened despite inotropic support and ECLS was initiated. He was relisted for transplantation, but another heart did not become available. A sacral decubitus ulcer developed, along with an elevated white blood cell count and mild renal failure. He was no longer believed to be a good transplant candidate and ECLS was discontinued after 370 hours.

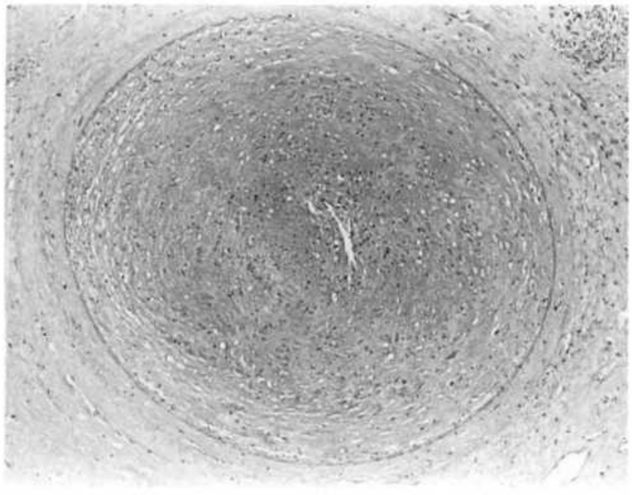

Fig 2. Representative area of left anterior descending coronary artery showing almost complete occlusion of the lumen by intimal proliferation. Focal lipid droplets are noted closest to the internal elastic lamina. (Hematoxylin and eosin, ×83 before 29% reduction.)

The third patient, a 6-year-old with tricuspid atresia and double-outlet right ventricle, was listed for transplantation and placed on ECLS after severe heart failure developed 6 weeks after a Fontan procedure. A chest tube in the right pleural space was removed during ECLS in an attempt to prevent any infectious complications that could preclude transplantation. Within 36 hours a right hemothorax developed, necessitating placement of another chest tube. Bleeding continued, and a thoracotomy was performed. The patient's respiratory and renal function deteriorated shortly thereafter, and the child was removed from the transplant list and ECLS was discontinued.

For all patients, flows ranged from 45 to 120 $mL \cdot kg^{-1} \cdot min^{-1}$ (mean, 70.4 $mL \cdot kg^{-1} \cdot min^{-1}$) and were maintained to provide adequate perfusion as demonstrated by mean arterial pressure, resolution of acidosis, increased urine output, and diminished need for inotropic support.

All patients were on multiple inotropes before initiating ECLS. The most commonly used inotropes included isoproterenol, dopamine, dobutamine, amrinone, and epinephrine. In 4 patients, including both survivors, all inotropes except low-dose dopamine were discontinued. In the other 2 patients the infusion rates were decreased substantially and at least one inotrope was discontinued. Both patients who were weaned from ECLS required inotropic support for 7 to 11 days after ECLS was discontinued.

In the 2 largest patients (>40 kg) the femoral artery and vein were cannulated. The remaining 4 patients had the right internal jugular vein cannulated for venous drainage. In 2 patients (weights, 37.5 and 22 kg) the oxygenated blood was infused through the femoral artery. Of the remaining patients 1 (14 kg) was cannulated through the right axillary artery and the smaller patient (4 kg) was cannulated through the right common carotid artery. The patients who were weaned from ECLS had their right internal jugular vein repaired. One survivor had the axillary artery repaired. The other surviving patient had her right carotid artery ligated, with no apparent neurological complications.

Hemofiltration was initiated for 4 patients, including both survivors, to facilitate additional fluid removal (see Table 1). No patient required hemodialysis.

Two patients received cyclosporin while on ECLS. The other transplant recipient was taken off cyclosporin while receiving OKT3. Cyclosporin doses ranged from 4 to 7 mg/kg. The cyclosporin levels did not appear to be affected by the ECLS circuit. The OKT3 level was also unaffected by the ECLS circuit, as evidenced by a lack of T cells on flow cytometry.

Mechanical complications included 1 case of hemolysis that resolved after a centrifugal pump was changed to a roller pump. Roller pumps were used for all other patients and no other episodes of hemolysis occurred. Two oxygenators were replaced owing to leakage of blood at the gas outlet. Both oxygenators were changed without incident.

Major physiological complications are listed in Table 1. The complications of pulmonary edema, sacral decubitus ulcer, and hemothorax after chest tube removal have been mentioned. Three patients had bleeding at the cannulation sites and required exploration. In each case a minor subcutaneous vessel was cauterized and no further bleeding developed. One patient sustained an irreversible neurological injury that developed 100 hours after ECLS was started. The cause of the injury was unknown. Permission for autopsy was denied. The patient was removed from circulatory support after neurological examination and electroencephalogram confirmed the absence of brainstem activity.

Comment

Mechanical circulatory support has been used to support patients with end-stage heart failure while a donor heart is sought [2–4]. Temporary mechanical support of the failing transplanted heart has also been described [5, 10]. At the present time mechanical support of pediatric patients is limited to intraaortic balloon pumping or ECLS. Intraaortic balloon pumping supports approximately 40% of left ventricular function. The access is simple and the cost moderate. Intraaortic balloon pumping is useful for moderate cardiac failure isolated to the left ventricle, but provides no right ventricular or pulmonary support. Extracorporeal life support can support 100% of cardiac and pulmonary function. Access is usually through extrathoracic vessels. Hemofiltration, if necessary, is easy to insert in the ECLS circuit and can allow full parenteral nutrition without fear of fluid overload. The disadvantages of ECLS include the need for anticoagulation, cost, and labor intensity.

This report suggests that ECLS may be useful for support of the transplanted heart with right ventricular failure shortly after implantation. Patients with an increased pulmonary vascular resistance greater than 8 Wood units are not usually considered candidates for orthotopic cardiac transplantation because it is expected that the right ventricle of the transplanted heart cannot respond to the sudden increase in afterload, especially after the ischemic period imposed by the transplant procedure [12]. Extracorporeal life support can temporarily provide cardiopulmonary support while the transplanted right ventricle recovers in patients with borderline elevation of pulmonary vascular resistance.

This report also suggests that a patient with biventricular failure due to severe acute rejection can be supported with ECLS to allow time for the antirejection therapy to become effective. Hemodynamic symptoms as a manifestation of rejection often do not develop in cardiac transplant patients receiving cyclosporin and prednisone until the hemodynamic status is so compromised that survival is in doubt. When rejection does not respond promptly to high-dose steroids, antithymocyte globulin, or OKT3, retransplantation has been thought to be the only effective option because antirejection therapy may require several days to effectively reverse rejection [13]. Extracorporeal life support may provide another option for managing the cardiac transplant patient with heart failure due to rejection.

Rogers and associates [11] have described the successful use of ECLS as a bridge to transplantation in a pediatric patient. Extracorporeal life support has also been used as a bridge to transplantation in adults, but the results have been disappointing [10]. In our experience a contraindication to transplantation developed during ECLS while waiting for a donor heart to become available. The difficulty in finding a pediatric donor heart resulted in a protracted length of time on ECLS. However, in all 3 cases an error in judgment or management may have contributed in part to the development of contraindications to transplantation. It may be illustrative to examine these points. In 1 patient (BF) a sacral decubitus ulcer and subsequent leukocytosis developed. Turning the patient more frequently may have prevented this complication. Another case (patient RS) was complicated by bleeding that developed after chest tube removal. A thoracotomy was required to control the bleeding and the patient was subsequently removed from the transplant list. In a patient awaiting transplantation the risk of infection from a chest tube in the pleural space must be weighed against the risk of manipulating a chest tube while the patient is anticoagulated. In the remaining patient (CG) complete cessation of left ventricular function developed within 12 hours after starting ECLS. Left-sided decompression by transatrial balloon and blade septostomy was not attempted until 36 hours later, by which time severe pulmonary edema had developed. Pulmonary edema resulting from a nonfunctioning left ventricle can pose a difficult problem. Extracorporeal life support does not completely decompress the left side of the heart in patients with an intact septum. Decompression procedures such as blade or balloon septostomy have been used with varying degrees of success. Transsternal placement of ECLS cannulas can allow better decompression, but this may also lead to bacterial contamination or infection of the mediastinum, making the patient an unacceptable candidate for transplantation. Despite our discouraging experience, ECLS may still have a role as a bridge to transplantation. However, complications may develop that could make the patient a poor transplant candidate.

Ethical questions have been raised regarding the use of ECLS or other mechanical support devices in conjunction with transplantation [14]. One author [15] has questioned whether transplantation (or retransplantation) should be performed in a suboptimal recipient given the scarcity of donor hearts. Answers to this and other difficult questions are not readily available. Nevertheless, this report suggests that ECLS can support the pediatric transplant patient with right ventricle failure due to pulmonary hypertension or biventricular failure due to rejection. Furthermore, it may be possible to provide cardiopulmonary support for pediatric transplantation. However, complications from ECLS may preclude subsequent transplantation.

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Pulmonary Resection Combined With Cardiac Operations

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Surgical management of patients with concomitant critical cardiac disease and resectable lung lesions is controversial. During a 7-year period (1982 to 1988), 21 patients underwent combined cardiac and pulmonary operations. Patients had cardiac symptoms only; the lung lesions were found on preoperative chest roentgenograms. The pathological diagnosis was established in only 2 of the patients before operation. All underwent concurrent pulmonary resection during cardiac operations requiring extracorporeal circulation. The pulmonary operations included 17 wedge resections and four lobectomies. The final diagnoses in 8 patients with stage I non-small cell lung cancer included epidermoid carcinoma (4), adenocarcinoma (3), and bronchoalveolar carcinoma (1). Postoperatively, 1 patient required a permanent pacemaker and 1 patient died. The actuarial survival at 5 years for all patients who underwent combined procedures was 95%. The 5-year survival for the 8 patients with lung cancer was 88% compared with 100% for those with benign pulmonary pathology ($p = 0.172$). This experience suggests that combining pulmonary resection with cardiac operations is safe and offers a favorable prognosis to a select group of patients.

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The discovery of an asymptomatic pulmonary lesion in a patient immediately before an open heart operation often poses a diagnostic and therapeutic problem. In part, this is due to the controversial issue as to whether one or two operations (combined) should be performed or postponed altogether [1, 2]. Usually, these patients have a surgically correctable cardiac disorder and are found to have an associated pulmonary process on a preoperative chest roentgenogram, often a solitary lung nodule. Because unrecognized or untreated lung cancer obviously alters a patient's long-term prognosis after a cardiac operation, if one is to consider performing these operations at the same time, it is important to understand the impact of such combined procedures on survival. Treatment of both diseases at the same time may be preferable provided it can be performed safely with the likelihood of good long-term results. The purpose of this review was to examine our experience with patients who were found to have a lung lesion just before a scheduled cardiac operation and their outcome when the combined approach was used at the State University of New York at Buffalo.

Material and Methods

The clinical records of 5,835 patients who underwent a cardiac operation requiring cardiopulmonary bypass at the Buffalo General Hospital between January 1982 and December 1988 were reviewed. Of these, 21 patients underwent combined pulmonary and cardiac procedures. The records of these patients were studied to assess presenting preoperative signs and symptoms, chest roentgenographic findings, diagnostic studies, indications for operation, operations performed, postoperative complications, the operative mortality (defined as death within the first 30 days after operation), and long-term outcome. Asymptomatic patients who had known synchronous problems with lung and heart disease were excluded from the study.

Perioperative data were obtained from the patients' hospital records. Follow-up information was collected through direct patient contact, from the patient's personal physician, or by telephone interview with surviving patients or family members.

Data are presented in the text and tables as simple percentage and frequency distributions. Values are expressed as the mean ± standard error of the mean. Patient survival is expressed by actuarial analysis according to the method of Kaplan and Meier and plotted at annual intervals using the day of operation as the starting time [3, 4]. Overall significance of values was determined by the approach of Lee and Desu [5]. Differences resulting in a p value of less than 0.05 were considered significant.

Results

The ages of the 21 patients (17 male, 4 female) with concomitant heart and lung diseases ranged between 38 and 75 years, with a mean of 60 ± 2.1 years. All patients had angina pectoris or dyspnea related to their heart

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Table 1. Cardiac Operations Performed in Patients With Asymptomatic Lung Lesions

Operation	No. of Patients
CABG	18
Aortic valve replacement and CABG	2
Mitral valve replacement	1
Total	21

CABG = coronary artery bypass grafting.

disease; the lung lesions were detected on the routine preoperative chest roentgenogram. Sixteen patients had a substantial history of smoking. With respect to cardiac pathology, the majority had coronary artery disease (85.6%) and the remainder had valvar heart disease (14.4%) (Table 1). Preoperatively, all patients were in New York Heart Association class III or IV. The pulmonary lesion was identified on the preoperative chest roentgenogram and appeared as a solitary nodule in 15 patients and an infiltrate in 6. At the time of operation, the pulmonary diagnosis was established in 2 patients and was unknown in 19 patients. The diagnosis was confirmed by preoperative bronchoscopic biopsy in 1 patient and by needle aspiration in the other 1. Pulmonary resections were performed through a median sternotomy incision. Lateral extension of the incision was not necessary in any of the patients. Standard pulmonary techniques were employed. Mediastinal and hilar lymph node sampling via median sternotomy was performed in patients in whom a diagnosis of lung carcinoma was established on frozen section. This was accomplished in the absence of anticoagulation.

Benign pulmonary pathology accounted for 61.9% of the lesions, 38.1% were bronchogenic carcinoma, and none were metastatic in origin (Table 2). In 19 patients, the diagnosis was established intraoperatively by simple wedge resection and frozen section analysis. This permitted an accurate histological diagnosis and confirmation of tumor-free margins in 17 patients (81.0%). Four patients (19.0%), however, required lobectomies (Table 3). Two of these were performed after intraoperative diagnostic wedge resections. In the other 2 patients, the diagnosis of lung cancer was made preoperatively. Lobectomies in these 4 patients were performed because of inability of obtaining tumor-free margins with a lesser resection.

All of the 8 patients with bronchogenic carcinoma were men and the mean age was 64 ± 1.5 years (range, 58 to 70 years). Of these, 6 patients underwent coronary artery bypass grafting, 1 underwent mitral valve replacement, and 1 underwent both aortic valve replacement and myocardial revascularization. The final pathological diagnoses included epidermoid carcinoma (4), adenocarcinoma (3), and bronchoalveolar carcinoma (1). The clinical and surgical stage was the same in all of these patients (see Table 3).

Pulmonary procedures were performed before bypass in 12 patients (57.1%) and after bypass in 9 (42.9%), during which time anticoagulation with heparin had not been instituted or had been reversed with protamine sulfate. Survival was not affected by performing pulmonary resections before bypass or after bypass ($p = 0.3898$). The pulmonary resections were considered appropriate for the given pathological condition in that limited wedge resections were performed for benign disease and definitive wedge resections or formal lobectomies were performed for bronchogenic carcinoma.

There were two postoperative complications. One occurred in a 72-year-old man who underwent aortic valve replacement, two-vessel coronary artery bypass grafting, and concomitant right upper lobectomy for squamous cell carcinoma. This patient required insertion of a permanent pacemaker for complete heart block. The second complication occurred in a 67-year-old man who underwent four-vessel revascularization and concomitant right upper lobectomy for squamous cell carcinoma. In this individual a sternal dehiscence developed that resulted in mediastinal infection and sepsis. He died on the twenty-ninth postoperative day, which accounts for the only 30-day hospital death in the series.

Follow-up data were available in all patients and averaged 43.5 months, ranging between 1 and 84 months. There were no late adverse postoperative sequelae from the resections. Of the 8 patients who underwent resection for bronchogenic carcinoma, 6 are currently alive. These patients are free of recurrent malignant disease at postoperative intervals ranging from 13 to 84 months. Only 1 patient who underwent lobectomy died of recurrent distant metastatic carcinoma 77 months after the original operation. Observed survival rate at 5 years for all patients who underwent combined procedures was 95% (Fig 1). This compared favorably with the 88% survival for patients with bronchogenic carcinoma and the 100% 5-year survival of 13 patients with benign pulmonary pathology. Survival probabilities for the cancer and the non-cancer patient groups were not significantly different ($p < 0.172$).

Comment

The natural history of patients with both treatable heart disease and lung cancer is unclear. Although a few

Table 2. Lung Pathology in 21 Patients

Final Diagnosis	No.	Percent
Benign	13	61.9
Granuloma	3	14.3
Hamartoma	3	14.3
Chronic inflammation	3	14.3
Sarcoidosis	1	4.8
Bronchiectasis	1	4.8
Bullae	2	9.5
Malignant	8	38.1
Bronchogenic carcinoma	8	38.1

Table 3. Operative Variables in Patients With Lung Cancer

Patient No.	Cell Type	TNM[a]	Tumor Location	Procedure
1	Epidermoid Ca	T1 N0 M0	R lower lobe	Wedge resection
2	Epidermoid Ca	T1 N0 M0	L upper lobe	Wedge resection
3	Epidermoid Ca	T1 N0 M0	R upper lobe	Lobectomy
4	Epidermoid Ca	T1 N0 M0	R upper lobe	Lobectomy
5	Adenocarcinoma	T1 N0 M0	R lower lobe	Wedge resection
6	Adenocarcinoma	T1 N0 M0	R lower lobe	Lobectomy
7	Adenocarcinoma	T1 N0 M0	R lower lobe	Lobectomy
8	Bronchoalveolar Ca	T1 N0 M0	L lower lobe	Wedge resection

[a] Clinical and surgical stage was the same in all patients.

Ca = carcinoma.

studies have addressed the natural history issue, it is difficult to separate the course of the cardiac disease from that of the associated pulmonary process [2, 6–8]. The fact that the surgeon rarely encounters these diseases simultaneously in the same patient probably reflects thorough preoperative evaluation before hospitalization. Even if the patient is found to have both diseases, there is a reluctance to combine a pulmonary resection with a heart operation for several reasons: (1) concern with potential technical problems associated with lung resection in the setting of anticoagulation, (2) limited exposure obtained via the median sternotomy incision, and (3) increased risk of mediastinal and pleural infection secondary to contamination in a patient whose immunologic response may be impaired owing to the effects of cardiopulmonary bypass.

A solitary peripheral lung mass in the symptomless patient frequently represents a relatively early primary carcinoma of the lung. Because a third of circumscribed solitary pulmonary nodules and two-thirds of radiologically indeterminate solitary pulmonary nodules are malignant, watchful waiting often is not considered a reasonable option in certain clinical situations. Ideally, all bronchogenic carcinomas should be removed without watchful waiting. Theoretically, bronchogenic carcinomas metastasize at some time, and watching even a small lung cancer may carry high risk for the individual patient. There are no certain radiographic features that can be considered absolutely diagnostic of lung cancer. In evaluating a peripheral lung nodule, a standard history, a physical examination, and laboratory and roentgenographic studies should be obtained. Cytologic examination of the sputum should be made although the results in patients with such lesions are less rewarding. Bronchoscopy is optional in the diagnostic evaluation as the yield is less than 10% for malignant nodules smaller than 2.0 cm in diameter [9]. Despite the advancements in preoperative biopsy techniques, many patients (approximately 20% to 25%) undergo lung operations without a histologic diagnosis. Thus, exploratory thoracotomy remains one of the most efficient means of diagnosis, particularly in those patients who have a resectable tumor. In the patient who has advanced heart disease and a lung nodule, one may prefer to avoid an intensive effort to establish a tissue diagnosis preoperatively if the patient has insufficient cardiac reserve and needs an urgent coronary or valvar heart operation.

Overall prognosis for all patients with carcinoma of the lung remains poor and the survival rate at 5 years is less than 10%. Complete surgical resection remains the most effective treatment for patients with non-small cell tumors who have stage I disease. The controversy regarding the optimal management of asymptomatic pulmonary lesions in cardiac patients scheduled to undergo an open heart operation mainly is centered around the timing of operation, ie, should the operations be performed at the same time or staged. The choice should be determined by the physiologic status of each given patient and the topographic extent of the tumor. The presence of cardiac risk

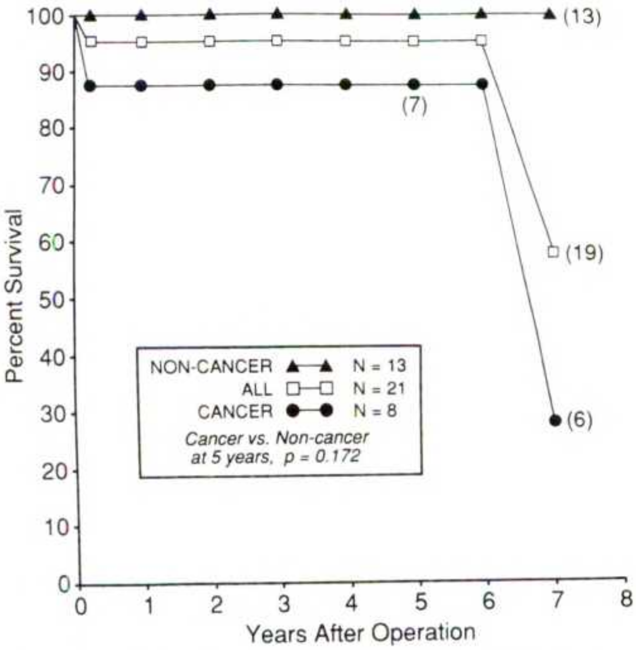

Fig 1. Survival curves of patients undergoing combined operations. Number of patients alive at specific time point is shown in parentheses.

factors, severity of symptoms, functional limitation, presence and degree of left ventricular dysfunction, age, and medical therapeutic options all are important factors that influence management.

Usually the cardiac operation dictates the operative approach. Although a median sternotomy incision is routine for almost all open heart procedures, it is not routine for pulmonary operations. This approach has been advocated for synchronous resection of bilateral lung lesions, particularly in the patient with severely impaired lung function [10, 11]. Resection of the lung lesion may be enhanced if a double-lumen endotracheal tube is used. Obviously, cardiac instability during insertion may preclude its use. In our series, all patients were extubated within 36 hours and no patient experienced intrapulmonary hemorrhage. There were no instances of prolonged air leak, excessive chest tube drainage, or pleural space complications.

If the patient's condition is stable, a wedge resection of the lung lesion with sufficient margins can be safely performed. While waiting for the histologic diagnosis, the cardiac operation can be completed. Should the diagnosis of lung carcinoma be made, mediastinal exploration and lymph node sampling should be performed after reversal of anticoagulation. Mediastinal lymph node sampling could be performed through a median sternotomy, although there is no question that complicated hilar and mediastinal dissections are more easily performed through a posterolateral thoracotomy. A potential limitation of resection for cancer through a median sternotomy is the relative inaccessibility of the posterior mediastinum for lymph node sampling. Although incomplete lymph node sampling could underestimate the extent of disease, the transsternal approach does not inherently compromise long-term survival. In patients with small peripheral lesions and without lymph node involvement (T1 N0 M0) a limited resection may be adequate treatment. As reported by Read and associates [12], a limited resection, when properly applied, is an acceptable option in patients with peripheral lung cancer. A lobectomy is indicated when a lesser resection will not completely remove the lung lesion. In our series, a wedge resection was insufficient in 4 patients. These were central lesions and precluded wedge excision. However, a limited wedge resection was diagnostic and resulted in satisfactory extirpation of the lesion in the majority of the patients (81%).

Recently, the Lung Cancer Study Group [13] reported a 5-year disease-free survival of 65% for surgically treated patients with T1 N0 M0 non-small cell cancer. In our series, disease-free survival of 87.5% at 5 years was achieved using the combined approach in patients with surgically and pathologically staged T1 N0 M0 non-small cell lung carcinoma. The observation that T1 N0 M0 patients who undergo conservative resection tend to die of their comorbid disease [12] also supports the view that performing both operations at the same time may be beneficial. Our experience suggests that combining pulmonary and cardiac procedures is appropriate and safe in select patients and is associated with good long-term outcome. The advantages of this approach include an immediate and definitive treatment of both conditions, the sparing of these patients from a second operation with its associated morbidity, and achievement of favorable survival.

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Antimicrobial Prophylaxis for Open Heart Operations

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Between 1986 and 1988, 450 adults undergoing coronary artery bypass, cardiac valve replacement, or both were enrolled into a prospective, randomized, comparative trial of cephalothin versus cefamandole as perioperative prophylaxis. They were assessed during their hospitalization and at 6 weeks and 6 months after discharge for postoperative infectious complications. Eleven patients had major postoperative infections including 5 with sternal wound infections (three bacteremic), 6 with bacteremia, 1 with prosthetic valve endocarditis, and 3 with severe venous donor graft site infections. Eight major infections occurred in patients receiving cephalothin prophylaxis and three in patients receiving cefamandole, with all five sternal wound infections occurring in the cephalothin group. Postoperative pathogens responsible for the major infections included gram-negative aerobes in 5 patients, *Staphylococcus aureus* in 4, and *Staphylococcus epidermidis* in 2. Preoperative colonizing staphylococcal isolates were not predictive of postoperative staphylococcal pathogens. Although there was no statistically significant difference in rate of major postoperative infectious complications using either cephalothin or cefamandole prophylaxis, there was a trend in favor of cefamandole. Gram-negative aerobes are becoming increasingly important pathogens in this setting.

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Antimicrobial prophylaxis for open heart operations is a well-accepted practice despite a paucity of supportive data documenting its value [1]. Postoperative infectious complications occurring in this setting are associated with substantial morbidity and mortality [2, 3] as well as cost [4]. These factors have been important in continuation of the practice and relevant in the search for the "ideal" prophylactic agent associated with the lowest postoperative infection rate. Despite numerous trials comparing a variety of agents [5–8] no clear conclusion can be drawn regarding the optimal choice. Recommendations at present are that a first- or second-generation cephalosporin or vancomycin be used [9].

At the University of Alberta Hospitals, cloxacillin had been used as the standard perioperative prophylactic agent for open heart operations until 1982 when it was replaced by cephalothin. A retrospective chart review of serious infectious complications of open heart operations including bacteremia, carditis, and sternal wound infections from January 1981 to November 1982 [10] revealed an infection rate of about 4%, with no significant difference in infection rates with either cloxacillin or cephalothin prophylaxis. This review prompted our development of a prospective, randomized trial of antimicrobial prophylaxis for open heart operations.

Prophylaxis refers to the prevention of infection caused by or related to a surgical or other invasive procedure. In terms of the infections for which one is attempting to provide antibiotic prophylaxis in open heart operations the most important are endocarditis and wound infection, primarily of the chest. Whether or not wound infection of the venous graft donor site should also warrant consideration for prophylaxis is a moot point as its consequences are generally less serious.

Pathogens most prominent in serious postoperative infections after open heart operations are staphylococci, with *Staphylococcus epidermidis* representing a clinically significant pathogen, particularly in prosthetic valve endocarditis [11, 12], but also in sternal wound infections [13]. Methicillin-resistant strains of *Staphylococcus aureus* and *S epidermidis* are recognized pathogens in this setting [12, 14, 15] and more difficult to treat by virtue of a more limited antibiotic armamentarium. Gram-negative aerobes [10, 16] are increasingly frequent pathogens, possibly related to prophylaxis directed primarily at gram-positive organisms, increasing age and debility of operative candidates, and tertiary care preoperative and postoperative environments.

Cefamandole is unique among the cephalosporins in that it has superior in vitro [17, 18] and in vivo [19] efficacy against coagulase-negative staphylococci, including methicillin-resistant ones. Its consideration as a prophylactic agent in open heart operations relates to this as well as its good bone [20], plasma, atrial muscle, and cardiac valve

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tissue [21] penetration. In rabbit models of endocarditis, there have been conflicting results regarding its efficacy in preventing experimental *S epidermidis* endocarditis [22, 23].

With this background information we developed a prospective, randomized triple-arm trial of antimicrobial prophylaxis comparing our standard agent, cephalothin, to cefamandole, to combined vancomycin and gentamicin. After 9 patients had entered into the study clinically significant preoperative hypotension developed in a patient receiving preoperative vancomycin prophylaxis. This generated considerable concern regarding the safety of vancomycin use in an anesthetic setting, particularly in patients with cardiac compromise. After a review of the literature related to cardiac events occurring in association with vancomycin administration during induction of anesthesia [24–26], the vancomycin/gentamicin arm was dropped. The study was revised to compare cephalothin and cefamandole perioperatively and is herein described.

The objectives of this study were to compare the efficacy of cephalothin to cefamandole in the prevention of infections after open heart operations, to examine the pathogens involved in serious postoperative infections, and to examine the relationship between preoperative colonizing staphylococci and pathogenic staphylococci producing serious postoperative infections.

Material and Methods

Study Protocol

The University of Alberta Ethics Review Committee for Human Experimentation reviewed and approved the study protocol in July 1985.

All patients older than 18 years undergoing an elective open heart operation for coronary artery bypass grafting or cardiac valve replacement or both at the University of Alberta Hospitals between April 1986 and October 1988 were considered for this randomized, comparative trial of cefamandole versus cephalothin perioperative prophylaxis. Patients were approached for consent if they met the following entry criteria: no history of allergy to penicillins or cephalosporins, not pregnant or lactating, serum creatinine level less than 150 μmol/L, no current infection, and no antibiotic therapy within 72 hours of operation. Four hundred fifty patients agreed to participate in the trial. Patients were assigned by a random number table to receive intravenously 2 g of either cephalothin or cefamandole preoperatively and every 6 hours for 48 hours postoperatively. All patients were operated on by one of five cardiovascular surgeons (J.C., E.G., D.M., P.P., L.S.).

Preoperatively, patients had routine hematologic and biochemical screening, a urinalysis, urine culture, and cultures of anterior nares, groin, and chest wall. All patients had blood cultures after bypass. Other postoperative cultures were obtained if clinically indicated. All patients underwent a standard surgical skin preparation with povidone-iodine (Betadine) preoperatively and intraoperatively. Body hair was clipped either the night before or on the morning of operation.

During their hospitalization, patients were assessed daily by one of three research assistants regarding the state of their operative wounds and any evidence of infection. Patients were also reviewed at 6 weeks and 6 months postoperatively. For patients unable to personally appear for a follow-up appointment, phone calls and a mailed questionnaire (Appendix 1) were used to determine their status after discharge.

Although surgeons were not blinded to study drug, the primary investigator who ultimately evaluated and confirmed all major infections was blinded to the prophylactic therapy at the time of randomization.

Postoperative infections were considered in three categories: major infections, minor infections of chest and leg, and secondary infections including respiratory tract, urinary tract, and miscellaneous infections. Major infections were defined as bacteremia, endocarditis, or deep sternal wound infections. Bacteremia was defined as positive blood cultures in the clinical setting of infection. Endocarditis was diagnosed on the basis of positive blood or tissue cultures in conjunction with clinical findings and echocardiographic studies or histologic confirmation. Deep sternal wound infections included mediastinitis and sternal osteomyelitis requiring both surgical intervention and antibiotic therapy. Sternal infections that responded to a short course of antibiotics alone were considered minor chest infections. Major leg infections were those requiring greater than 1 week of hospitalization or additional operation for debridement or plastic surgical repair. All major infections were bacteriologically confirmed.

A minor wound infection of the leg or chest was defined as a wound characterized by localized erythema, redness, and warmth in association with either a positive culture or physician-initiated antibiotic therapy.

Respiratory tract infections were defined as purulent sputum in association with chest roentgenographic abnormalities or pathogens in sputum culture and physician-initiated antibiotic therapy. Urinary tract infection was defined as more than 10^8 organisms/L (10^5/mL) or symptomatic disease with 10^6 organisms/L (10^3/mL) and physician-initiated antibiotic therapy. Miscellaneous infections included chest tube site infections, phlebitis, and stitch abscesses.

Statistical Analysis

The study was designed with a fixed sample size of 500 and an interim analysis at the midpoint of the study. Because of economic and time constraints, the study was terminated prematurely when 450 patients had been entered.

Analysis was conducted on patients enrolled on an intent-to-treat basis with patients who met the entry criteria being evaluated for efficacy and safety. Postoperative follow-up was obtained for all patients. The data were coded on a format developed through a pilot project. Revisions were made over the course of the study in this format. Data were cleaned both by the coder scanning the data and by the nursing and medical staff. All major and minor infections were reviewed by the principal investigator. Cleaning was also done via the computer by limits set on key variables. Descriptive analyses and frequencies were produced on all variables. SPSS was the software employed in the analysis.

All baseline demographic, clinical, and preoperative variables were compared between the two groups by χ^2, Fisher's exact test, and t tests to determine if there were differences between treatment groups at baseline. If a difference in a key variable was found at baseline, adjustments were to be employed using the key variable as a covariate in the analysis of covariance. This adjustment was not required because there were no significant differences at baseline. Efficacy and adverse effect comparisons by patient and by adverse effect counts are calculated by χ^2 and binomial tests.

In all statistical tests the level of significance was set at p less than 0.05, 2-tailed.

Results

Table 1 outlines the demographics of the populations randomized to each drug and demonstrates no significant difference in age, sex, surgical procedure, length of preoperative hospitalization, presence of chronic illness preoperatively, duration of operation, duration of bypass, or number of days on a ventilator. Variables not listed in the table, but for which there was no statistical difference between the two groups, included body weight, duration of prophylaxis, and surgeon distribution. Seventy-seven percent of both groups underwent coronary bypass grafting, whereas 18.7% of the cefamandole group and 20.1% of the cephalothin group underwent cardiac valve replacement. Four percent of the cefamandole and 2.7% of the cephalothin group underwent combined coronary artery bypass and cardiac valve replacement. The majority of patients (310/362) undergoing coronary bypass procedures underwent a single internal mammary artery implant with only 7 having bilateral internal mammary artery implants. Forty-five had vein grafts. Thirty-three percent of the cefamandole group and 37% of the cephalothin group had chronic illnesses preoperatively including diabetes mellitus, chronic obstructive pulmonary disease, hypertension, arthritis, thyroid disorders, peripheral vascular disease, obesity, cerebrovascular disease, diverticular disease, reflux esophagitis, and peptic ulcer disease.

Table 1. Demographic Information and Medical Background by Group

	Test Medication		
Variable	Cefamandole (N = 224)	Cephalothin (N = 224)	p Value
Age (y)	57.8 ± 9.4	57.2 ± 11.0	NS
Sex			
Male	168 (74.7%)	182 (81.2%)	NS
Female	57 (25.3%)	42 (18.8%)	
Basis for operation			
Coronary artery bypass	174 (77.3%)	173 (77.2%)	
Cardiac valve replacement	42 (18.7%)	45 (20.1%)	NS
Both	9 (4.0%)	6 (2.7%)	
Time in hospital preop (d)	2.2 ± 1.9	2.5 ± 2.0	NS
Chronic illnesses			
Yes	75 (33.3%)	83 (37.1%)	NS
No	150 (66.7%)	141 (62.9%)	
Duration of operation (min)	282.0 ± 70.5	281.3 ± 71.4	NS
Time on CPB (min)	129.9 ± 39.3	130.9 ± 41.0	NS
Time on ventilator (d)	2.0 ± 1.4	1.9 ± 1.3	NS

CPB = cardiopulmonary bypass; NS = not significant ($p > 0.05$).

Table 2. Postoperative Infections by Group

	Test Medication		
Variable (Frequency)	Cefamandole	Cephalothin	p Value
Major infections (11)			
Sternal wound (5)	0	5	NS
Bacteremia (2)	1	1	NS
Endocarditis (1)	1	0	NS
Leg (3)	1	2	NS
Minor infections (96)			NS
Leg (65)	36	29	
Chest (22)	8	14	
Chest and leg (9)	4	5	
Secondary infections (53)			NS
Respiratory (19)	11	8	
Urinary tract (18)	8	10	
Miscellaneous (29)	14	15	

NS = not significant ($p > 0.05$).

At the conclusion of the trial in May 1989, 445 patients were considered evaluable, 5 having been excluded because of the presence of preoperative infections. Two of these 5 patients had been randomized to receive cefamandole and 3 to receive cephalothin.

Postoperatively 11 patients were considered to have had major infections, 96 patients had minor infections, and 53 patients had secondary infections. The frequency of major, minor, and secondary infections by treatment is detailed in Table 2. There were no significant differences in frequency of major infections between the two groups, with three in the cefamandole group and eight in the cephalothin group. By infection, there were 5 patients with major sternal wound infections (all in the cephalothin group), 2 patients with bacteremia (one in each group), 1 patient with endocarditis (in the cefamandole group) and 3 patients with major leg infections (1 in the cefamandole and 2 in the cephalothin group). The mean hospital stay of patients with major infections was 50.0 ± 7.6 days for the cefamandole group and 34.8 ± 25.5 days for the cephalothin group (not statistically different).

Minor infections shown in Table 2 were identical in frequency, with 48 in both groups and no statistical difference between the groups.

Secondary infections included respiratory, urinary tract, and miscellaneous infections. Their frequencies of occurrence are also shown in Table 2 and demonstrate no statistical difference between the cefamandole and cephalothin groups. The miscellaneous group of the secondary infections included 9 patients with chest tube site infection, 9 with phlebitis, 5 with stitch abscess, and 1 patient each with prostatitis, cellulitis, pharyngitis, pseudomembranous colitis, pacemaker wire infection, and vaginitis.

Of the 11 patients with major infections, 5 had severe sternal wound infections requiring surgical intervention. Three of these 5 patients were also bacteremic. Six patients had clinically significant bacteremia including the 1 patient with endocarditis. Three patients had major leg wound infection. Four patients had more than one major infection. The details of these 15 major infections in the 11 patients are presented in Table 3.

Review of the microbiology of the major infections demonstrates that in 4 patients methicillin-sensitive *S aureus* was the primary pathogen. Coagulase-negative staphylococci were primary pathogens in only 2 patients with major infections, 1 a case of prosthetic valve endocarditis and the other a case of sternal osteomyelitis. The latter infection was due to methicillin-resistant coagulase-negative staphylococci. In 5 of the 11 patients with major infections, gram-negative aerobes were considered primary pathogens and usually were present with other organisms, including other gram-negative aerobes (in 3 of 5) or enterococci (in 3 of 5).

Of note, in all but 1 of the cases of major infection the onset of signs and symptoms was within 2 weeks of operation and confirmed bacteriologically as early as 3 days postoperatively. One case of sternal osteomyelitis due to coagulase-negative staphylococci (patient 238) was not diagnosed until 8 weeks postoperatively although the patient had been febrile and received antibiotic therapy for superficial venous graft site infections within a week of his operation.

Comparing the group of patients with and without major infections, there were no significant differences noted in age, sex, procedure, duration of operation, length of preoperative hospitalization, surgeon distribution, or antibiotic prophylaxis. Similarly, in examining the minor leg and chest infections, no difference was noted in the above-mentioned variables between patients with and without postoperative infections. The presence of chronic illness preoperatively was not statistically associated with major postoperative infection risk though specific diseases were not examined in detail. There was no statistically significant increase in risk of major infection in patients with insulin-dependent diabetes.

Of the 10 patients with major infections who had undergone coronary bypass grafting, 8 had had a single left internal mammary graft and 1, bilateral internal mammary artery grafting. Internal mammary grafting was not significantly associated with likelihood of a major infection in this study.

Mortality rate was 4% (17/445) within 6 months of operation with 2/17 (11%) of these deaths occurring in patients with major infection. One of these two deaths was definitely related to infection. There was no significant difference in the prophylaxis these 17 patients received, with eight deaths in the cephalothin group and nine in the cefamandole group.

Potential drug side effects including nausea, vomiting, diarrhea, and bleeding disorders were examined and showed no significant difference between the group receiving cephalothin and the group receiving cefamandole.

Examination of the preoperative staphylococcal isolates of patients with major postoperative staphylococcal infections (Table 4) showed no clear relationship between preoperative isolates from anterior nares, chest, or groin and postoperative pathogen. In 2 of 4 cases *S aureus* was isolated preoperatively and postoperatively with both preoperative and postoperative isolates showing the same sensitivity pattern. In the other 2 cases postoperative *S aureus* infection was not associated with preoperative *S aureus* colonization. In the last two of six postoperative staphylococcal infections, the postoperative pathogen was coagulase-negative staphylococcus whereas preoperative colonization involved coagulase-negative staphylococcus strains showing different sensitivity patterns.

Comment

This study, although demonstrating no significant difference in infection rates after open heart operations with either cephalothin or cefamandole prophylaxis, highlights a number of important issues. Perhaps the most important of these are the principles of prophylaxis well summarized and discussed by Platt [27]. One should clearly define the infections one is attempting to prevent, determine the most likely pathogens, and then select the safest, least toxic, and least costly agent that optimally will cover these organisms. If these requirements cannot be met, the virtues and disadvantages of the agents must be balanced against the relatively low risk (generally accepted as less than 5%) of a potentially serious, even life-threatening infection after a clean operation.

Even though vancomycin and gentamicin cover the most likely possible pathogens (*S aureus* and *S epidermidis* as well as aerobic gram-negative organisms) and might represent reasonable choices in established or likely infection after open heart operations, they should not be considered reasonable routine prophylaxis in this subpopulation because of potential toxicities and ecologic costs. The literature [24–26] and our own experience document a risk of arrhythmias and hypotension with perioperative vancomycin use. In patients with cardiac compromise the potential risks of using this agent on a routine basis probably outweigh the potential benefits. Additionally, both vancomycin and gentamicin have renal and ototoxicity [28], which together could be additive. This broad-spectrum combination also raises serious concerns about the promotion of resistance and possible superinfection with enterococci or yeast. The monetary cost of vancomycin/gentamicin prophylaxis would be about 8 times as much as that of the standard cephalothin regimen this

Table 3. Major Infectious Complications of Open Heart Operations

Patient No.	Procedure	Infection	Organism Isolated	Sensitivities	Prophylaxis	Predisposition	Outcome
002	CABG × 3	Bacteremia	*S aureus*	S to Ox/K/M	K	Bilateral internal mammary implants	Surgical debridement; chronic oral antibiotics; sternal wire removal 1 year after discharge
		Deep sternal wd (osteomyelitis)	*S aureus*	S to Ox/K/M			
046	AVR	Bacteremia Endocarditis	*S epidermidis*	S to Ox/K/M	M	Current smoker	6 w IV antibiotics
087	CABG × 1 + MVR	Bacteremia	*S aureus*	S to Ox/K	K	Current smoker, COPD	Sternal dehiscence; radical sternal debridement; death within 6 mo
		Deep sternal wd (mediastinitis)	*S aureus*	S to Ox/K			
132	CABG × 5	Deep sternal wd (mediastinitis)	*Pseudomonas aeruginosa*		K	Current smoker, obesity, prolonged postop ventilation	Sternal debridement × 3; multiple muscle flap grafts
			Enterococci	S to Amp/V			
163	CABG × 3	Bacteremia	*Klebsiella oxytoca*	S to K/G	K	Current smoker, prolonged postop ventilation	Death
189	CABG × 4	Venous graft site	*Klebsiella pneumoniae*	S to K/G/M	M	Obesity, Crohn's, >5 days preop hospitalization	Surgical evacuation left thigh hematoma + 6 w IV antibiotics; readmission 1 month after discharge with streptococcal cellulitis right venous graft site
			E coli	S to K/G/M			
			Proteus mirabilis	S to Amp K/G			
			Enterococci				
			Bacteroides fragilis				
200	CABG × 3 + AVR	Bacteremia	*Streptococcus faecalis*	S to Amp/V	K	Electrocardioversion 4 days postop	Sternal dehiscence; sternal debridement & 4 w IV antibiotics
		Deep sternal wd (mediastinitis)	*Enterobacter aerogenes*	R to K			
			Morganella morganii	R to K			
			E coli	S to K/G			
238	CABG × 5	Deep sternal wd (osteomyelitis)	Coagulase-negative staphylococcus	S to V/G R to K	K	IDDM, obesity	Sternal curettage & 6 w IV antibiotics
269	CABG × 4	Venous graft site	*S aureus*	S to Ox/K	K		Readmission 9 days after discharge for 2 w IV antibiotic
417	CABG × 4 + ventricular aneurysm resection	Bacteremia	*S aureus*	S to Ox/K/M	M	>5 days preop hospitalization	6 w IV antibiotics
444	CABG × 3	Venous graft site	*Klebsiella oxytoca*	S to G R to K	K	Current smoker, COPD, IDDM, >5 days preop hospitalization	16 d IV antibiotics
			Pseudomonas aeruginosa	S to G			

Amp = ampicillin; AVR = aortic valve replacement; CABG = coronary artery bypass grafting; COPD = chronic obstructive pulmonary disease; G = gentamicin; IDDM = insulin-dependent diabetes mellitus; IV = intravenous; K = cephalothin; M = cefamandole; MVR = mitral valve replacement; Ox = oxacillin; R = resistant; S = sensitive; V = vancomycin; wd = wound.

Table 4. Major Staphylococcal Infections After Open Heart Operation

Patient No.	Pathogen	Sensitivities	Preop Colonizing Isolates	Sensitivities
002	*Staphylococcus aureus*	S to Ox/E/K/M; R to Pen	Groin, nose, chest: *S aureus*	S to Ox/E/K/M; R to Pen
046	*Staphylococcus epidermidis*	S to Ox/E/K/M; R to Pen	Groin: CNS	S to K/M
			Chest: CNS #1	R to K/M
			CNS #2	S to K/M
087	*Staphylococcus aureus*	S to Ox/E/K; R to Pen	Chest: CNS; Groin: CNS	
238	CNS	S to V/G; R to Pen/Ox/E/K	Chest: CNS	S to Ox
269	*Staphylococcus aureus*	S to Ox/E/K; R to Pen	Chest: CNS	
417	*Staphylococcus aureus*	S to Ox/E/K/V/M; R to Pen	Nose: *S aureus*	S to Ox/M

CNS = coagulase-negative *Staphylococcus*; E = erythromycin; G = gentamicin; K = cephalothin; M = cefamandole; Ox = oxacillin; Pen = penicillin; R = resistant; S = sensitive; V = vancomycin.

institution had been using (assuming nine 2-g doses of cephalothin at about $37 Canadian [Cn] versus seven 1-g doses of vancomycin and four 70-mg doses of gentamicin at about $300 Cn). Thus, what might appear one of the most efficacious choices would not necessarily be the safest, least toxic, or least costly and thus is far from ideal for prophylaxis in this setting.

First- and second-generation cephalosporins are reasonable considerations for prophylaxis in this setting. Though one might consider both cephalothin and cefamandole to have a similar gram-negative spectrum, cefamandole's spectrum is extended beyond cephalothin's, and there is well documented in vitro activity against *S epidermidis* with cefamandole [17]. In our study, there was no significant difference in efficacy between cephalothin and cefamandole in preventing postoperative infections, nor was there a difference in the incidence of side effects. There was, however, an obvious trend in favor of the cefamandole arm with all cases of severe sternal wound infection occurring in the cephalothin arm. Examining economics, the cost of cefamandole is some three times that of cephalothin ($122.00 Cn versus $37.00 Cn). Whether the costs of five sternal wound infections would counterbalance the increased cost of the drug if these infections could have been prevented is an unanswered question. Are the drug's potential benefits worth the increased cost?

Recognizing cefamandole's in vitro superiority against *S epidermidis*, could there be an explanation for the lack of statistical difference in infection rates in this study? Perhaps in view of the low overall infection rate of 2.5%, 445 patients and 11 major infections were not enough to demonstrate a difference between these two agents. It has been stated that some 4,953 patients would be needed to detect a true significant difference between documented rates of infection of about 4% with first-generation cephalosporins and about 3% with second-generation cephalosporins at a power of 80% [29]. Clearly our study had too few patients to reach this level of statistical power. Based on the rate of major infections that occurred, the power of this study to detect a 50% difference in treatments was only 55%.

If indeed cefamandole's superiority lies in its improved activity against *S epidermidis*, it is probable that there were not enough infections due to this organism to demonstrate any difference. *S epidermidis* was the primary pathogen in only 2 of the 11 patients with major infections in this study.

Might it be that the specific agent used is of secondary importance and that an increased awareness and concern about infection led to improved infection control techniques and procedures? Certainly the 2.5% incidence of major infections in this study is an improvement over the previously documented retrospective incidence at this institute of 4.1% [10]. Ferrazzi and associates [30] showed that improving the surgical and operating room protocols as well as increasing awareness of the dangers of infection can reduce the incidence of infections after cardiac operations.

Is our prophylaxis in part contributing to the changing characteristics of postoperative infections? In this study, five of the 11 major infections, the majority, were due to gram-negative aerobes. By providing good gram-positive coverage, have we contributed to the development of postoperative gram-negative infections? By providing improved gram-negative coverage in our choice of perioperative prophylaxis might we be promoting the development of enterococcal and fungal infections postoperatively? Should we consider narrower spectrum therapy directed at gram-positive organisms rather than broader coverage?

Most of the major postoperative gram-negative infections were leg infections, and one wonders whether prophylaxis directed against them is reasonable and cost effective. Certainly they are not as serious as the other major infections, but they may be associated with increased costs related to prolonged hospital stay and antibiotic use. It is noteworthy that 79 of 445 patients, almost 16% of all study patients, received antibiotic therapy for major and minor venous graft site infections.

A number of factors have been shown to predispose to surgical wound infections after cardiac operations. These include obesity, diabetes, length of preoperative hospitalization greater than 5 days, and current smoking [31]. A history of chronic obstructive pulmonary disease, prolonged bypass pump time, lengthy duration of operation, and surgical reexploration [32] have also been demonstrated to be important risk factors. Culliford and coworkers [33] found a greater incidence of mediastinitis after internal mammary artery mobilization and this was particularly notable with bilateral artery implants. Loop and colleagues [34], on the other hand, found bilateral internal thoracic artery grafting in nondiabetics carried no greater risk of wound complication than vein grafts or single artery grafts. In reviewing our 11 cases of serious infection, at least one of these "risk" factors was identified in the majority of patients.

Knowledge of preoperative colonization by *S aureus* unfortunately does not appear to predict either postoperative pathogens or likelihood of infection. This study confirmed our previous findings [35] and prompted the discontinuation of our previous practice of routine preoperative culturing of anterior nares, chest, and groin in our institution.

Finally, one should remember that prophylaxis is not 100% effective. Our 11 major infections confirm that despite using agents known to be effective against recognized pathogens, postoperative infections cannot always be prevented. The safest, least toxic, most effective against likely pathogens, and least costly agents should be selected whenever possible. This randomized, comparative trial of cefamandole and cephalothin did not show a statistical difference in efficacy, toxicity, or side effects. Whether cefamandole's theoretically superior in vitro activity against *S epidermidis* activity outweighs its increased cost remains uncertain and may need further examination. The numbers of patients, however, required to show statistically significant differences between agents achieving rates of postoperative infection less than 5% are enormous.

In summary, cefamandole is a reasonable choice as a prophylactic agent for open heart operations as far as efficacy, toxicity, and infection rates are concerned. In this study it appeared to be superior to cephalothin though the differences in infection rates failed to achieve statistical significance. The entire issue of prophylaxis for open heart operations is complex and poses many questions, few of which have been answered by this study. Rather, some have been clarified and confirmed as important areas for future investigation.

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Appendix 1

Name: ______________________ No: __________

MONTHLY QUESTIONNAIRE

OPEN HEART SURGERY PROTOCOL

Please indicate if you have any of the following during the past month:

	YES	NO
Fevers	___	___
Chills	___	___
Weight loss	___	___
Sweats	___	___
Diarrhea	___	___
Burning on urination	___	___
Foul-smelling urine	___	___
Cough (if so, are you coughing up any abnormal secretions?)	___	___
Shortness of breath	___	___
Redness or drainage from incision site	___	___
Any infection diagnosed by your family doctor	___	___

If "YES" to any of the above, we suggest you contact a physician and indicate below the site of infection and treatment, if known.

____

____

____

Please return it to us, in the stamped, self-addressed envelope provided by ______________________________.

If you have any questions or concerns, please do not hesitate to contact us.

Clinical Research Nurse
Division of Infectious Diseases
Walter Mackenzie Centre
University of Alberta Hospitals
Edmonton, Alberta

Phone: ______________________________

Tension Pneumopericardium as a Complication of Single-Lung Transplantation

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Tension pneumopericardium is distinctly uncommon in the adult population. We present a case of tension pneumopericardium as a complication of lung transplantation in a 54-year-old woman with thromboembolic pulmonary hypertension who underwent single-lung transplantation.

(Ann Thorac Surg 1990;50:808–10)

Simple tension pneumopericardium most commonly is seen in infants requiring mechanical ventilation [1]. In adults, the entity is rare, with only 5 adult cases in an extensive 1984 review [1]. We report a case of tension pneumopericardium in a lung transplant patient.

The patient was a previously healthy woman who first noted in 1983, at the age of 49 years, the onset of vague chest pain and decreasing exercise tolerance. An outside hospital evaluation in April 1984 revealed multiple perfusion defects on a ventilation-perfusion scan, normal collagen vascular disease studies, and room air blood gases as follows: arterial oxygen tension, 76 mm Hg; arterial carbon dioxide tension, 31 mm Hg; pH, 7.46. Heart catheterization revealed a pulmonary artery pressure of 106/32 mm Hg and showed no septal defects or mitral valve abnormalities. The patient was placed on continuous anticoagulation therapy, but was felt not to be a candidate for thromboendarterectomy after a pulmonary angiogram revealed multiple small-vessel obstructions.

The patient's first evaluation at the University of Mississippi Medical Center was in July 1988, at which time her exercise tolerance was limited to approximately one-half block. An echocardiogram revealed a moderately dilated, poorly contractile right atrium and right ventricle, mild tricuspid and mitral regurgitation, and minimally decreased left ventricular function. On gated heart scan, the left ventricular ejection fraction was 0.69 and the right ventricular ejection fraction was 0.43. Pulmonary function testing revealed a forced expiratory volume in 1 second that was 82% of predicted with forced vital capacity of 91% of predicted. Bilateral ascending venograms were negative. It was believed that she suffered from thromboembolic pulmonary hypertension.

On September 1, 1989, a lung became available for transplantation from a 16-year-old girl who suffered closed-head trauma. The lung was transported by cold immersion with a total ischemic time of 4½ hours. Cardiopulmonary bypass was required secondary to intolerance of pulmonary artery clamping, but left lung transplantation with omental wrap was performed without difficulty. Pulmonary artery systolic pressure dropped from 130 to 70 mm Hg immediately after transplantation. On arrival at the surgical intensive care unit, mean arterial blood pressure was 77 mm Hg, pulmonary artery pressure was 45/28 mm Hg, and on 1.0 inspired oxygen fraction, 7½ cm H_2O positive end-expiratory pressure, 25 cm H_2O pressure control, and rate of 12/min, the patient's blood gases were: arterial oxygen tension, 65 mm Hg; arterial carbon dioxide tension, 46 mm Hg; and pH, 7.27.

Postoperatively the patient was given azathioprine, antithymocyte globulin, and intravenous infusion of prostaglandin E_1 with subsequent administration of cyclosporine. The patient was weaned to 0.6 inspired oxygen fraction, but on postoperative day 4 apparent rejection developed with diffuse infiltrates on the left, fever, and worsening of oxygenation and ventilation. A methylprednisolone bolus resulted in a prompt improvement in the respiratory status with a decrease in the inspired oxygen fraction from 1.0 to 0.6 and an increase in arterial oxygen tension from 51 to 69 mm Hg. However, the patient's condition subsequently deteriorated with the development of enterococcal sepsis, renal failure, hepatic failure, and worsening of her respiratory status unresponsive to a second treatment of methylprednisolone. On postoperative day 14, the patient's hemodynamic status worsened and a pneumopericardium was noted on chest radiograph (Fig 1). At this time, the patient was requiring 12.5 cm H_2O positive end-expiratory pressure and 38 cm H_2O pressure control resulting in a peak inspiratory pressure of approximately 50 cm H_2O. A 16-gauge angiocatheter was introduced into the pericardial sac by the subxiphoid approach with release of air and the following changes: pulmonary capillary wedge pressure, from 20 to 10 mm Hg; central venous pressure, from 18 to 8 mm Hg; pulmonary artery pressure, from 56/24 to 65/26 mm Hg; blood pressure, from 76/36 to 110/40 mm Hg; cardiac output, from 5.0 to 6.6 L/min; mixed venous oxygen

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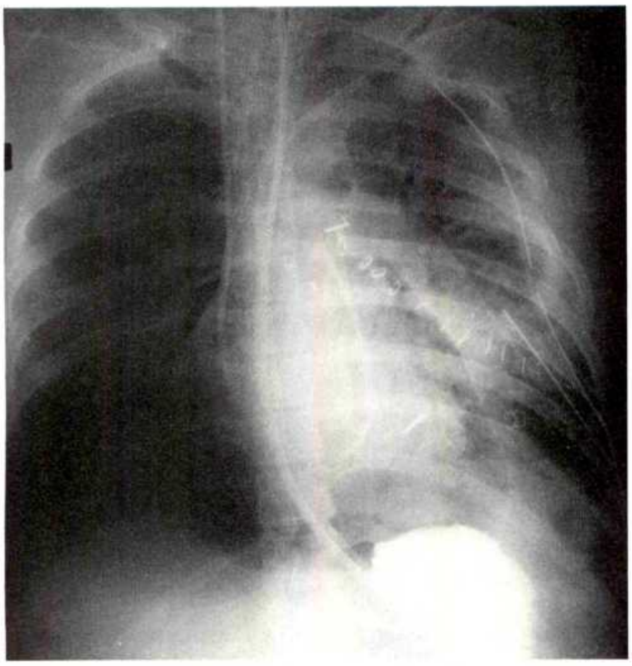

Fig 1. Chest radiograph showing pneumopericardium. Note that the pericardial sac on the left is outlined by the omentum brought into the chest.

saturation, from 50% to 58%; and arterial oxygen tension, from 57 to 62 mm Hg. A chest radiograph (Fig 2) showed resolution of the pneumopericardium. Air reaccumulated over several hours and a 24F chest tube was placed into the pericardial sac through the subxiphoid approach. Multiorgan failure continued with the addition of disseminated intravascular coagulation and worsening sepsis. The patient died on postoperative day 16 after prolonged hypoxemia and hypotension despite 1.0 inspired oxygen fraction and maximal vasopressor administration. Autopsy revealed fibrinous pericarditis, a small (0.15 mm) focus of separation at the bronchial anastomosis, and multiple thromboemboli throughout the arterial tree of the right lung but not the donor left lung. There was also diffuse alveolar wall damage with hyaline membrane formation that was present bilaterally but that was more severe in the donor lung.

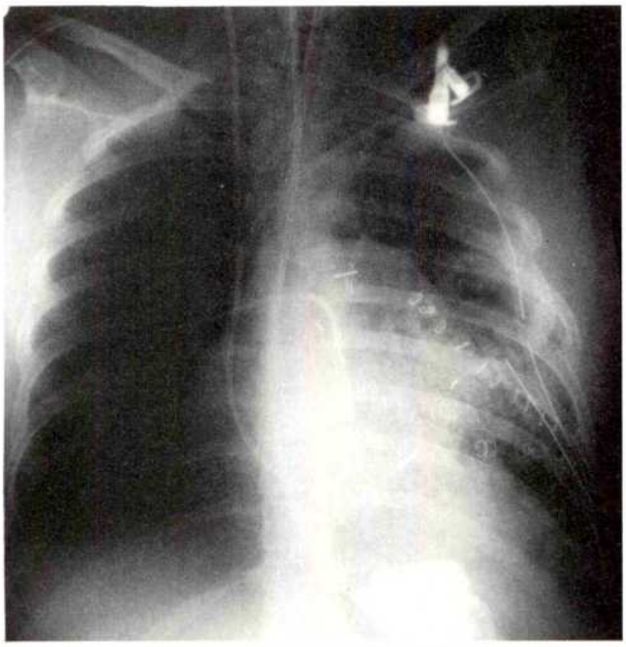

Fig 2. Chest radiograph immediately after aspiration of the pericardial air.

Comment

The first description of a pneumopericardium was in 1844 by Bricheteau [2]. A pneumopericardium may be simple (air-containing only) or complicated (air- and fluid-containing) and, additionally, may or may not result in cardiac tamponade. In contrast to infants, a tension pneumopericardium in adults is a rarely reported entity. In the review by Cummings and associates [1] of 252 patients with pneumopericardium, there were 81 cases of simple tension pneumopericardium with adults accounting for only 5 of the 81 cases. Since this review in 1984, there have been at least 20 cases reported of simple tension pneumopericardium in adults—9 cases secondary to blunt chest trauma [3–7], 5 cases secondary to penetrating chest trauma [8–12], 4 postoperative patients (2 after esophagogastrectomy, 1 after pneumonectomy, 1 after tracheostomy) [13–16], 1 ventilated asthmatic [17], and 1 case during an endotracheal laser operation [18].

Signs in tension pneumopericardium may include muffled heart sounds, bradycardia, pulsus paradoxus (or absent pulses), increased central venous pressure, and hypotension [1]. The electrocardiogram may show low voltage or bradycardia [1]. In addition to air limited by the pericardial sac, the radiograph may also reveal a diminishing size of the heart if tamponade is developing [5].

A pneumopericardium may be the result of direct communication with the pericardium (as in fistulas or penetrating injuries) or may result from alveolar rupture (as in blunt injury or in patients on ventilators). With alveolar rupture, air can dissect along the perivascular sheath to the mediastinum and then into the pericardium [4]. Our patient was at increased risk of alveolar rupture owing to high positive end-expiratory pressure and peak inspiratory pressure. Air that reached the mediastinum may have more easily entered the pericardial sac, which had been violated both during removal of the diseased lung and during reanastomosis of the donor lung. Additionally, the omental wrap around the bronchial anastomosis was in close proximity to the pericardium and may have contributed to the tension pneumopericardium through a ball-valve effect.

Heart-lung transplantation has been the traditional operation for patients with pulmonary hypertension and cor pulmonale. However, there has been an increased interest in single-lung transplantation in these patients to increase donor availability and to hopefully decrease the occurrence of bronchiolitis obliterans. Resolution of pulmonary hypertension and right ventricular dysfunction has been demonstrated at long-term follow-up in patients who underwent thromboendarterectomy for thromboembolic pulmonary hypertension [19]. Additionally, there have been several successful single-lung transplantations for primary pulmonary hypertension. This includes a patient with a mean pulmonary artery pressure of greater than 60 mm Hg who after transplantation had a normal pulmonary artery pressure and right ventricular function at the time of outpatient catheterization (Cooper JD, personal communication, 1990).

Although there has been an increased number of reports in the last several years, a simple tension pneumopericardium is still a rare entity in the adult population.

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Vagal Schwannoma

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A patient with a benign encapsulated intrathoracic vagal schwannoma is presented and the literature is reviewed. The right paratracheal tumor was identified incidentally on a chest film and excised using a right thoracotomy. Although rare, vagal schwannomas should be considered for any mediastinal mass along the vagus nerves especially when the paratracheal tumor produces minimal or no respiratory symptoms suggesting a slow-growing, encapsulated process.

(Ann Thorac Surg 1990;50:811–3)

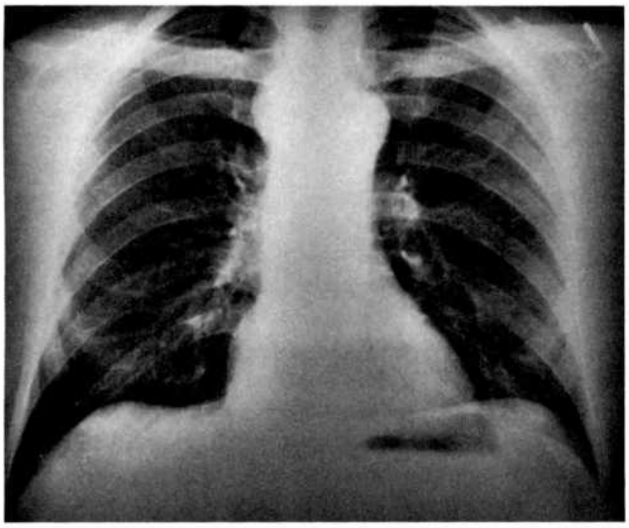

Fig 1. Posteroanterior chest roentgenogram.

Schwannomas (neurilemomas) are peripheral nerve sheath tumors. Although more common in the extremities, they may develop within the chest, where they usually originate in the posterior mediastinum from spinal nerve roots, intercostal nerves, or the sympathetic trunk. Most are benign, solitary, and asymptomatic. The diagnosis is suggested by radiographic identification of a tumor in the characteristic posterior mediastinal location. Treatment is resection to remove the tumor and confirm the diagnosis. Schwannomas arising from the vagus are rare. A patient with such a tumor is presented and the literature is reviewed.

A 63-year-old white man was admitted to the Baltimore VA Medical Center with left flank pain. The patient's symptoms promptly improved with bed rest and acetaminophen. Urologic and gastrointestinal evaluation were within normal limits. An admission chest film, however, revealed a right paratracheal mass (Figs 1, 2). The patient did not smoke, had been previously employed as a steelworker, and was active and healthy. His past medical history was remarkable for medically controlled hypertension. The patient denied any respiratory symptoms. There was no history of weight loss, fevers, night sweats, or other constitutional symptoms. The patient's only medication was captopril, 25 mg orally twice per day.

On physical examination there was no peripheral adenopathy, carotid bruits, cervical venous distention, or tracheal deviation. His voice was normal. The chest was clear, and the cardiovascular examination was within normal limits. The remainder of his physical examination was unremarkable. Laboratory screening including elec-

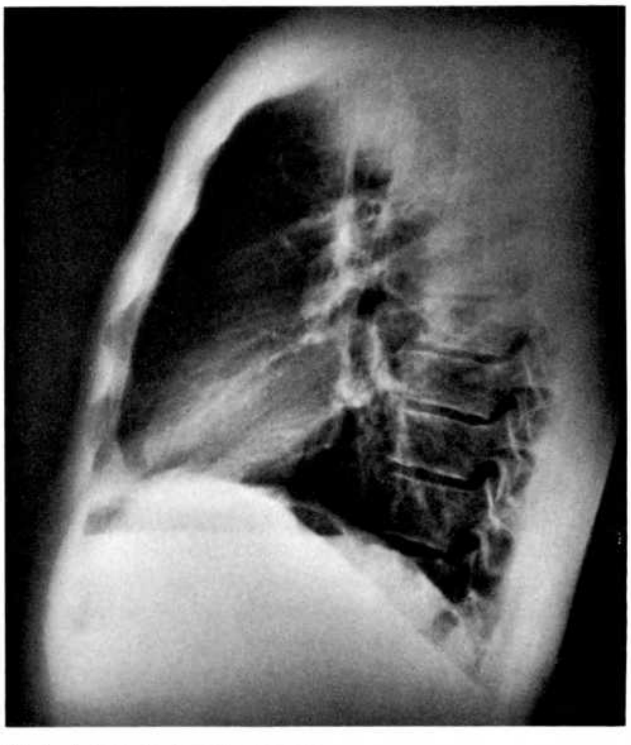

Fig 2. Lateral chest roentgenogram.

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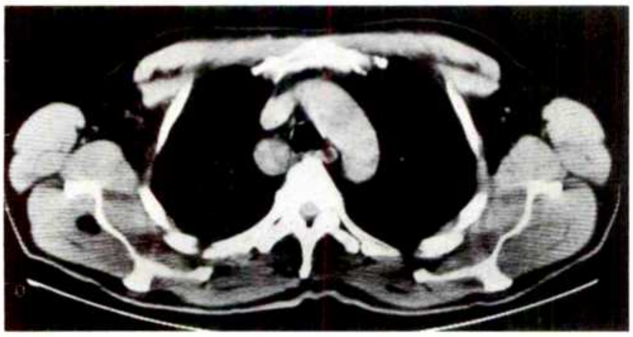


Fig 3. Chest computed tomographic scan cut through distal trachea showing mediastinal tumor.

trolyte levels, white cell count, hematocrit, and urinalysis were within normal limits.

Work-up included a negative complete head and neck evaluation, and a negative skin test with purified protein derivative. A chest and abdominal computed tomographic scan showed a 2 × 3 × 3-cm noncalcified, noncystic, homogeneous right paratracheal mass (Fig 3), which was interpreted as most suggestive of enlarged paratracheal lymph nodes. Bronchoscopy was negative, and at mediastinoscopy, a firm, encapsulated right paratracheal mass was seen. Biopsies of this mass returned schwannoma.

The patient was subsequently explored through a right fourth intercostal space thoracotomy. At exploration there was 2 × 3 × 3-cm rubbery, well-encapsulated mass adjacent to the mid-trachea. The fusiform mass was oriented along the path of the vagus nerve and appeared to arise from it. There was no right intrathoracic adenopathy, and no pulmonary lesions were identified. The mass was excised easily in conjunction with a segment of involved vagus nerve, and the patient had an uncomplicated postoperative course.

Final pathology confirmed a solitary benign encapsulated schwannoma (Fig 4).

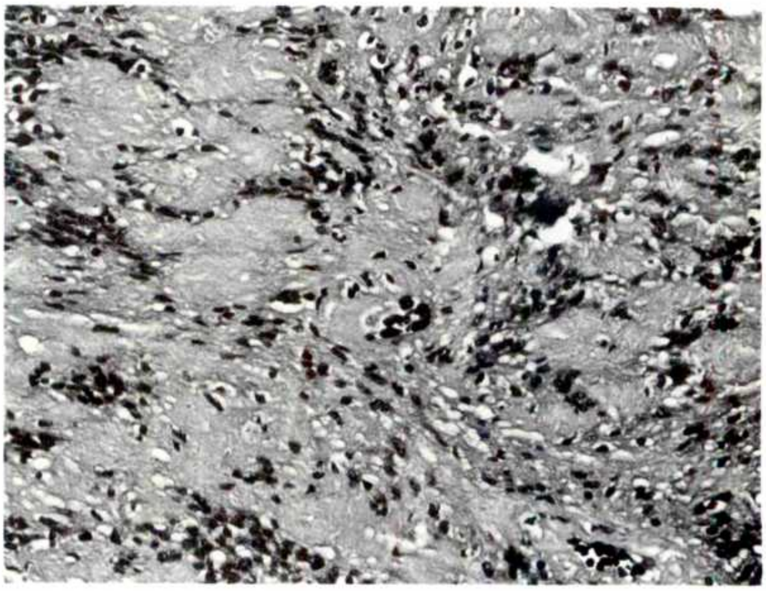
Comment

Schwannomas (neurilemomas) are peripheral nerve sheath tumors that can arise from any peripheral nerve or nerve root. Although most commonly found in the head, neck, or extremities, they also occur within the chest arising from the sympathetic chain, spinal nerve roots, intercostal nerves, brachial plexus, or phrenic or vagus nerves. Neurogenic tumors are the most common tumors of the posterior mediastinum, and the majority of these in adults are schwannomas and neurofibromas. Schwannomas are found in all age groups; there is a slight female preponderance. Most are benign and solitary, although multiple schwannomas have been reported [1]. Peripheral tumors are first identified as a painless mass, whereas intrathoracic tumors are usually identified incidentally on chest roentgenography as with our patient. When symptomatic, or if associated with von Recklinghausen's disease, malignant schwannoma should be suspected. Despite this patient's moderate-sized right paratracheal mass there were neither constitutional symptoms nor symptoms of bronchial irritation.

Schwannomas of the vagus nerve are particularly uncommon. Das Gupta and co-workers [2] identified 2 cases of vagal origin from a total of 303 benign solitary schwannomas. Whitaker and Droulias [3] reported 4 of 76 benign encapsulated schwannomas as arising from the cervical vagus. Combining the results from three large series of thoracic neural tumors [4–6] for a total of 214 tumors, there were only 3 cases of vagal schwannoma. Two additional patients had anterior mediastinal schwannomas in which the specific nerve of origin was not identified.

Treatment is resection both to remove the tumor and for

Fig 4. Section of benign schwannoma showing the characteristic Antoni A pattern with rows of nuclei in nuclear palisades and Verocay body formation (eosinophilic cell bodies nearly encircled by rows of nuclei). (Hematoxylin and eosin, ×290 before 13% reduction.)



diagnosis. Benign encapsulated tumors can be removed from peripheral nerves by enucleation. Some believe that the ability to enucleate these benign tumors is so characteristic that it distinguishes them from neurofibromas [3]. Others [4] have stressed that the histological and morphological differences between thoracic schwannomas and neurofibromas is less clear. In our patient the tumor was discrete and well encapsulated yet inseparable from the right vagus, and complete resection required removing a short segment of involved nerve.

Malignant degeneration of benign tumors has been described but is rare [2, 7]. For benign encapsulated schwannomas, resection is curative.

Although rare, vagal schwannoma should be included in the differential diagnosis of a mediastinal mass along the vagus nerves.

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Notice From the American Board of Thoracic Surgery

The part 1 (written) examination will be held at the Hyatt-Regency, Dallas Fort Worth Airport, Dallas TX, on February 16, 1992. The closing date for registration is August 1, 1991.

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Successful Fontan-Type Operation for a Nonresectable Right Ventricular Tumor

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A large intracavitary right ventricular tumor in a 24-year-old patient was considered nonresectable because it involved the interventricular septum, the free ventricular walls, and the tricuspid valve. Surgical palliation consisted of closure of the tricuspid and pulmonary valves, and the right atrium was anastomosed to the pulmonary artery bifurcation. The patient is asymptomatic 7 years after operation, and the neoplasm (a rhabdomyoma) has not increased in size.

(Ann Thorac Surg 1990;50:814–6)

Primary cardiac neoplasms are uncommon lesions. Surgical resection is indicated as soon as the diagnosis is made in all symptomatic patients, and whenever there is clinically significant tumor-related inlet or outlet obstruction [1]. Complete surgical resection is the treatment of choice, and most patients with benign cardiac tumors are probably cured by resection [2]. However, complete removal of rhabdomyomas is frequently impossible because the myocardium and the interventricular septum may be extensively involved [1]. A partial resection of the intracavitary obstructive portion of the tumor offers adequate palliation [1, 3]. Although wide resection and reconstruction of the atrial walls may be successfully accomplished [4, 5], severe damage to vital structures may occur when attempts are made to remove the entire tumor involving the ventricular myocardium and septum [3, 4]. In certain cases, valve replacement may be performed if the valve is destroyed by the tumor [4, 6]. To accomplish a complete excision for "nonresectable" cardiac neoplasms, heart transplantation has been performed in rare occasions [2]. We report a case of a modified Fontan procedure in a patient with a large right ventricular rhabdomyoma.

A 24-year-old woman had a history of dyspnea on exertion and syncopal episodes. Her blood pressure was 90/60 mm/Hg, her pulse rate was 110/min, and her respiratory rate was 28/min. A 2/6 mid-systolic murmur was heard, and marked jugular venous distention, hepatomegaly, and bilateral pretibial edema were noted. The electrocardiogram and chest roentgenogram were noncontributory. The diagnosis of a large right ventricular tumor was established with two-dimensional echocardiogram. Heart catheterization confirmed the above findings. The left ventricular pressure was 90/0–10 mm Hg; left ventriculogram revealed no obstruction in this chamber, and a competent mitral valve.

She underwent operation on April 30, 1982. Through a median sternotomy incision, cardiopulmonary bypass was established using bicaval cannulation and moderate hypothermia. Cold crystalloid cardioplegic solution was used for myocardial protection. After the huge right atrium was opened, the tricuspid valve was inspected and was found to be grossly involved with a large, firm, grayish-white mass that was occupying almost the entire right ventricular cavity. It was considered to be nonresectable because it was firmly attached to the interventricular septum, the free walls of the right ventricle, and the tricuspid valve (Fig 1). A biopsy specimen was obtained. No attempts were made to remove the tumor. It was decided to isolate the right ventricle and to bypass it, applying Fontan's principle. The tricuspid valve was closed with a Dacron patch, and the pulmonary valve was oversewn after transection of the main pulmonary artery. The pulmonary artery bifurcation was mobilized and brought behind and to the right of the ascending aorta. The incision in the pulmonary artery was extended into the right branch to achieve an atriopulmonary anastomosis as large as possible, as described by Kreutzer and associates [7]. Thus, the right atrial appendage was anastomosed directly to the pulmonary artery without interposition of a valve or a conduit. An anastomosis 7 cm in diameter was obtained (Fig 2).

Aortic clamping time was 43 minutes, and cardiopulmonary bypass was discontinued after 72 minutes. She remained hemodynamically stable, in normal sinus rhythm, with a central venous pressure from 10 to 14 cm H_2O. The postoperative recovery was uneventful, and she was discharged on the 11th postoperative day. Histological examination of the intraoperative biopsy showed classic "spider cells," characteristic of rhabdomyoma (Fig 3). These cells contain abundant glycogen stored in large vacuoles, which compress thin cytoplasmic strands extending from the nucleus toward the cell membrane, as described by Fenoglio and co-workers [8].

The patient was readmitted 1 week later because of rapid atrial fibrillation. After cardioversion, she was medicated during 1 year with digitalis. In April 1988, 6 years after operation, her left ventricular function remained normal by echocardiographic criteria (Fig 4). The size of

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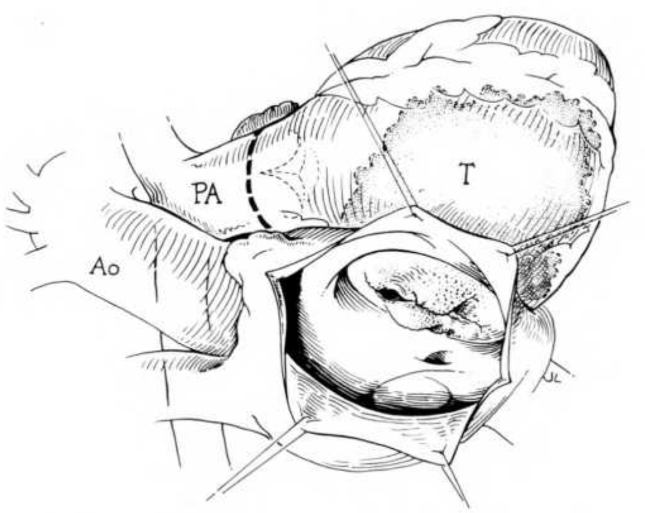

Fig 1. Surgical findings: the tricuspid valve was grossly involved with the tumor, as seen through the right atriotomy. The cannulas are not shown. (Ao = *aorta;* PA = *pulmonary artery;* T = *tumor.)*

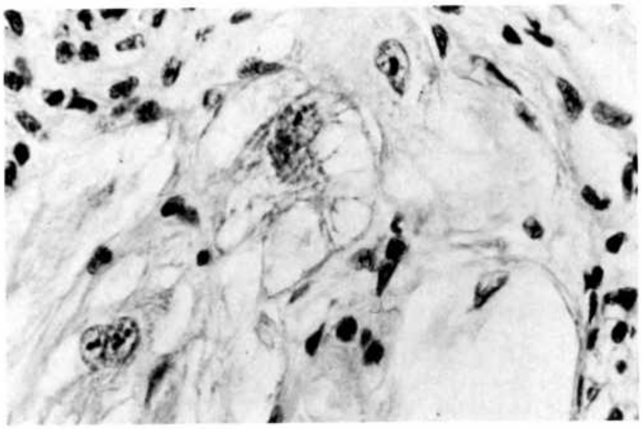

Fig 3. Photomicrograph showing typical "spider cells." (Hematoxylin and eosin, ×450 before 50% reduction.)

the right ventricular tumor and of the enlarged right atrium were similar to those seen in her preoperative echocardiogram. At the time of her last follow-up visit in March 1989, she remained asymptomatic and in normal sinus rhythm.

Comment

Cardiac rhabdomyomas are frequently multiple and may be intracavitary or intramural, involving the right or left ventricle, or both [1, 8]. Most of the patients are infants, in whom the mortality is 70%, usually caused by obstruction to blood flow [8]. An incomplete resection of the obstructive portion may be a life-saving procedure [1, 3], and long-lasting symptomatic relief up to 4 years has been reported [3]. These tumors do not show notable growth potential, and may even have some tendency to regress spontaneously [8, 9]. Tuberous sclerosis may coexist in approximately 50% of the patients with rhabdomyoma of the heart [1, 8], but was not present in our patient.

The excellent result obtained in this case suggests that the principle of Fontan can be safely applied when a right ventricular tumor is considered nonresectable. The prerequisites for employing the Fontan operation, outlined by Choussat and associates [10], are normal left ventricular function (end-diastolic pressure less than 12 mm/Hg), competent left atrioventricular valve, normal pulmonary vascular resistance (less than 4 U/m^2), pulmonary artery branches without obstruction, and normal sinus rhythm. These criteria were met in the case being presented.

The Fontan operation was initially designed to repair tricuspid atresia [11]. Modifications of the Fontan procedure have been employed successfully in a wide variety of complex congenital anomalies, including univentricular

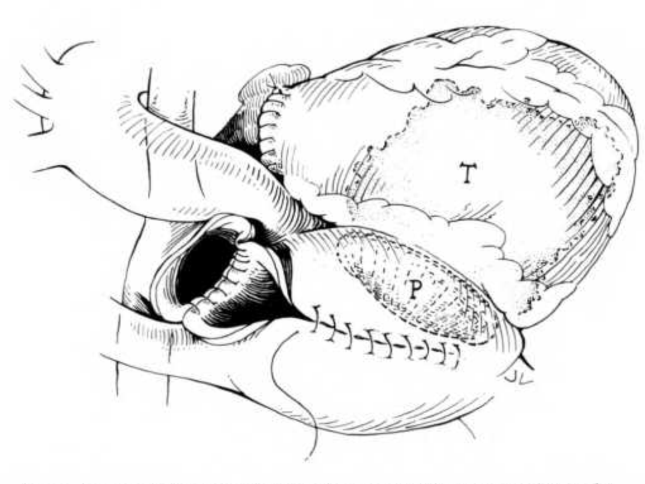

Fig 2. Surgical diagram showing the closed pulmonary valve and a patch covering the tricuspid valve. The direct anastomosis between the right atrial appendage and the pulmonary artery is shown before completion. (P = *patch;* T = *tumor.)*

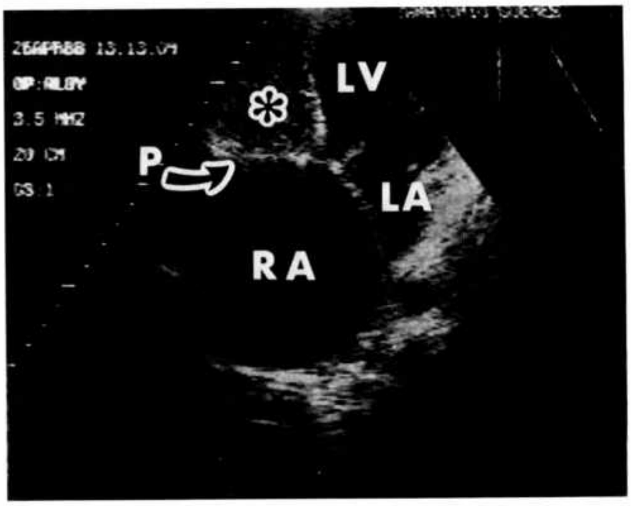

Fig 4. Postoperative echocardiogram showing the tumor (*) occupying most of the right ventricular cavity and the Dacron patch closing the tricuspid valve. (LA = *left atrium;* LV = *left ventricle;* P = *patch;* RA = *right atrium.)*

heart, and in lesions that were corrected by bypassing a right ventricle that was very small or hemodynamically inadequate [12]. We now report a successful Fontan-type operation with exclusion of a right ventricular neoplasm.

We are very grateful for the artistic illustrations by Joanie Livermore, and the echocardiograms and follow-up data obtained by Carlos Bruno, MD, and Rosa Villamayor, MD.

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Catastrophic Thrombosis of Porcine Aortic Bioprostheses

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Omar M. Lattouf, MD, Robert A. Guyton, MD, and Douglas C. Morris, MD

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Hemodynamically critical thrombotic stenosis of porcine bioprosthetic valves in the aortic position without thrombotic predisposition is rare. Two patients at our institution abruptly manifested thrombotic stenosis of porcine bioprosthetic valves in the aortic position within 3 months of implantation without apparent predisposing factors. Clinicians should consider this rare but catastrophic complication in the appropriate setting.

(Ann Thorac Surg 1990;50:817–9)

Porcine bioprosthetic cardiac valves have been used for over 20 years. In comparison with mechanical valves, they have a lower predilection for causing thromboembolism and a tendency in adults for gradual rather than abrupt catastrophic failure [1–3]. Bioprosthetic valvar dysfunction may involve calcification with stiffening or tearing of the leaflets, collagen disruption with leaflet tears, endocarditis, perivalvar regurgitation, fibrous overgrowth of the valve annulus and leaflets, leaflet lipid infiltration, leaflet hematoma, thromboembolism, thrombosis, and "stent creep" [1, 2, 4]. Thrombosis of bioprosthetic valves with resultant stenosis or occlusion most commonly occurs in the mitral position in the setting of low flow, left atrial enlargement, or atrial fibrillation [5, 6]. Thrombosis of porcine bioprosthetic valves in the aortic position without predisposing or precipitating cause is rare, and usually occurs more than 3 months after implantation [7–10]. Two patients recently were seen at our institution who suffered catastrophic thrombotic stenosis of porcine bioprosthetic valves in the aortic position less than 3 months after bioprosthesis placement without predisposing cause.

Patient 1

A 32-year-old man underwent aortic valve replacement with a standard Carpentier-Edwards 23-mm porcine bioprosthetic valve for severe aortic regurgitation secondary to pneumococcal endocarditis in late December 1988. His preoperative coagulation profile and platelet count were normal except for a mildly elevated bleeding time. Cardiac catheterization had revealed normal left ventricular systolic function. During the immediate postoperative period he was treated with intravenous penicillin. He was discharged 2 weeks later at his request and received 2 weeks of intravenous cefonicid followed by oral amoxicillin and clavulanate without anticoagulation or antiplatelet therapy. He did well until several hours before his death 10 weeks later, when he had abrupt onset of dizziness and dyspnea. He was hypotensive and dyspneic on arrival in the emergency room. Electromechanical dissociation developed shortly after presentation and he could not be resuscitated. Echocardiogram performed with the patient in extremus revealed severe, diffuse left ventricular systolic dysfunction and minimal motion of the aortic valve leaflets. Autopsy disclosed large amounts of fibrin material firmly adherent to the aortic surfaces of the bioprosthetic leaflets. A few neutrophils and lymphocytes were entrapped in the fibrin clot. Many neutrophils and an occasional eosinophil covered the surfaces of the fibrin clots. No microorganisms or inflammatory reaction were detected within the leaflets themselves. Two of the three valve leaflets had severely restricted motion and the valve orifice was slitlike (Fig 1). Postmortem blood cultures and a drug screen were unremarkable. Radiographic assessment of the bioprosthetic valves revealed no clinically significant calcification of the leaflets. Death was attributed to thrombosis of the bioprosthetic valves with resulting critical stenosis.

Patient 2

A 70-year-old man underwent aortic valve replacement with a standard Carpentier-Edwards 27-mm porcine bioprosthetic valve for severe aortic regurgitation in April 1988. His left ventricular ejection fraction was calculated to be 0.52 by preoperative cardiac catheterization. His preoperative coagulation profile, platelet count, and bleeding time were within normal limits. He remained in sinus rhythm throughout the perioperative period, and he was discharged without anticoagulation or antiplatelet therapy. He did well until 12 weeks later, when he experienced an episode of near-syncope accompanied by incontinence. He came to the emergency room alert and in no distress with a grade 3/6 harsh spindle-shaped murmur, but abruptly became hypotensive, dyspneic, cyanotic, and unresponsive. In this state no murmur was audible. He briefly required external cardiac massage. Echocardiography revealed diffuse left ventricular systolic dysfunction without demonstrable aortic bioprosthetic valve leaflet motion. The hemodynamically unstable patient underwent an emergency open-heart operation

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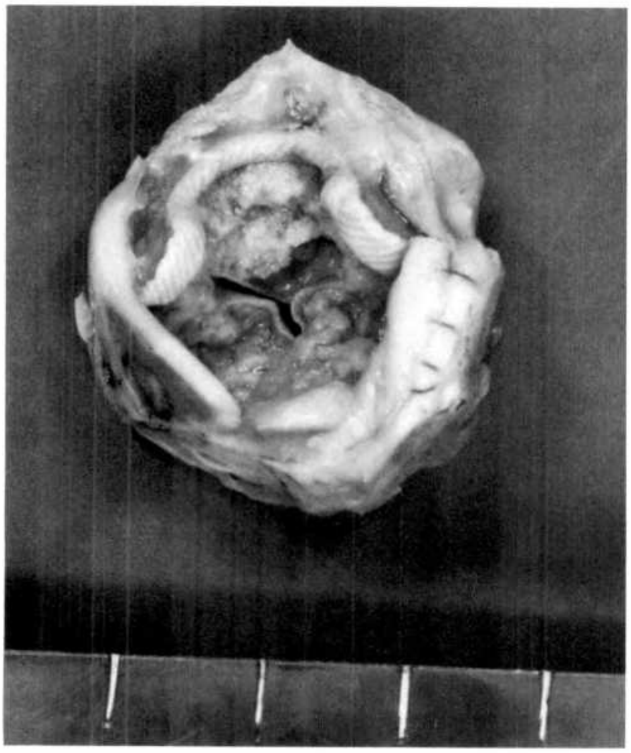

Fig 1. Aortic view of bioprosthetic valve removed during autopsy shows thrombotic material filling the aortic cusps (each demarcation on the straight edge equals one centimeter).

without further work-up. As suspected, a thrombosed porcine bioprosthetic valve was found and replaced with another bioprosthetic valve.

He was discharged receiving warfarin sodium (Coumadin) and has a normally functioning bioprosthetic valve at 10 months of follow-up. Pathological examination of the explanted bioprosthetic valve revealed all three bioprosthetic leaflets to be virtually immobile due to adherent thrombi, which were up to 8 mm in thickness and coated the aortic surfaces of the leaflets. Radiologic assessment of the explanted valve did not demonstrate any clinically significant leaflet calcification.

Comment

Thrombosis is an unusual cause of failure of porcine bioprosthetic valves [3, 11]. It most commonly involves bioprosthetic valves in the mitral position [5, 6]. Explanted and autopsy specimens of porcine bioprosthetic valves, however, frequently reveal the presence of fibrin or thrombi on leaflet surfaces. Most of these depositions are small and focal lesions that do not restrict leaflet movement [12].

Thrombi critically immobilizing leaflets of bioprosthetic valves in the aortic position and causing valve failure due simply to outflow obstruction are rarely reported. Zuhdi [11], Oyer [3], Thiene [5], Fishbein [13], and their associates reported cases of aortic porcine bioprosthetic valve thrombosis associated with low cardiac output, "stent creep," leaflet calcification, and estrogen use. Franken [7], Cohen [8], Croft [9], Lesnefsky [10], and co-workers published accounts of critical thrombotic stenosis of an aortic bioprosthetic valve without apparent predisposing factors occurring as early as 4 months after placement. The cases described here differ from these previous reports in the following ways: (1) both patients were asymptomatic until several hours before presentation, (2) manifestation of their bioprosthetic dysfunction was abrupt and catastrophic, (3) neither had apparent predisposing factors for valvar thrombosis, and (4) critical thrombosis occurred earlier in the postimplantation period. Conceivably other cases of unexplained sudden death after placement of bioprosthetic valves could be due to similar scenarios. Certainly one must consider valvar thrombosis in patients with bioprosthetic valves who acutely decompensate. Success with thrombolytic therapy of thrombosed mechanical valves has been reported, and this might be warranted in clinically stable patients [14]. With catastrophic valvar thrombosis, as described in the present 2 patients, we believe that given the uncertainty of success, the dire consequences of failure, and the added difficulty of operation after administration of a thrombolytic agent, thrombolytic therapy should be used only as a temporizing maneuver if operation is not immediately available. A short course of anticoagulation after bioprosthetic valve implantation regardless of position might prevent early catastrophic thrombosis in the aortic position. Given the very low incidence of this phenomenon, however, we do not think it prudent to chronically anticoagulate patients with bioprosthetic valves in the aortic position.

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REVIEW OF RECENT BOOKS

Medical Management of the Cardiac Surgical Patient

Edited by Richard J. Gray and Jack M. Matloff
Baltimore, Williams & Wilkins, 1990
378 pp, illustrated, $52.95

Reviewed by Richard S. Jackson, MD

Medical Management of the Cardiac Surgical Patient by Drs Gray and Matloff is a nicely pieced together and pragmatic guide to the perioperative care of patients with acquired surgical cardiac abnormalities. Initial sections deal concisely and practically with indications for cardiac surgery of acquired problems by revealing important influencing factors such as the Coronary Artery Surgery Study. Risk factors for perioperative mortality and morbidity are reviewed especially in a chapter devoted to operations in the elderly.

Central sections deal with preparation of the patient for the planned procedure, intraoperative management such as fluid and pharmacologic practices, and practical matters of perioperative and hemodynamic monitoring. A section on early postoperative recovery phase gives wide attention to surgical complications such as tamponade, bleeding, and perioperative myocardial infarction as well as medical complications including arrhythmias and pulmonary and renal complications and management. Cognitive and psychological changes around the time of operation as well as the nursing care of the cardiac surgical patient are topics that are not neglected in this text.

The authors are to be complimented for including discharge planning and late-phase follow-up sections that address short-term and longer term physical therapy recommendations, guidelines for dietary management, and risk factor modification.

Although conceptual and basic physiologic information is used concisely, the focus of the text is to provide its readers with a comprehensive, practical, and thoughtful review and discussion of a system of care of the patient with acquired cardiac surgical problems. The text should be highly useful to more senior medical students and lower and middle level resident staff and physician's assistants in providing them with a sound and manageable body of material upon which to base their involvement with the care of cardiac surgical patients. Additionally, members of intensive care nursing groups as well as other support groups involved with these patients would find much of the perspective provided helpful in rounding out their knowledge of these patients' course. Senior-level residents and attending staff can certainly find this text helpful for providing comparative guidelines in establishing and maintaining successful patterns of practice.

Burlington, Vermont

Combined Repair of Transposed Great Arteries and Total Anomalous Pulmonary Venous Connection

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We report an unusual association of transposition of the great arteries and total anomalous pulmonary venous connection in the right atrium that was successfully repaired by a modified Mustard procedure.

(Ann Thorac Surg 1990;50:820–1)

Transposition of the great arteries (TGA) combined with total anomalous pulmonary venous connection (TAPVC) is a very rare congenital cardiac anomaly. To our knowledge 2 cases of TAPVC associated with TGA have been previously reported. The first anatomical description of this combination, with all pulmonary veins entering the right atrium, was reported in 1933 [1]. The second report was on a 9-year-old girl with TGA and a supracardiac type of TAPVC [2] who underwent successful Mustard operation at the age of 16 years [3].

We report repair of TGA combined with TAPVC in the right atrium in a neonate.

A female neonate, 3 kg of body weight, was referred to our hospital because of a systolic ejection murmur discovered at birth. The family history was not relevant and no cyanotic spells were noticed. At physical examination all peripheral pulses were present and equal, respiratory and cardiac rates were increased, and hepatomegaly was noted. A chest roentgenogram showed an egg-shaped heart that was slightly enlarged. The superior mediastinum was also widened and the lung field appeared mildly congested. On two-dimensional echocardiography the diagnosis was situs solitus, levocardia, ventricular D-loop, d-TGA, atrial septal defect, ventricular septal defect, patent ductus arteriosus, and TAPVC in the right atrium. Cardiac catheterization and angiography performed 5 days later confirmed the diagnosis of TGA with TAPVC in the right atrium, but showed the presence of multiple ventricular septal defects with a gradient of 45 mm Hg across the left ventricular outflow tract. The catheterization data (Table 1) showed an oxygen saturation in the right atrium 18% higher than that in the inferior vena cava. The aortic and pulmonary artery saturation were almost identical. A pulmonary blood flow to systemic blood flow ratio (Q_P/Q_S) of 1.7:1 was calculated. Because of persistent signs of congestive heart failure despite medical treatment, the decision to operate was made when the patient was 2 months of age.

The surgical procedure was performed on conventional cardiopulmonary bypass and cardiocirculatory arrest. The right atrium was opened and the diagnosis was definitely cleared up (Fig 1): all four pulmonary veins were connected with the right atrium. A persistent left superior vena cava draining into the coronary sinus was also noted. A modified Mustard procedure was performed creating a tunnel around the orifices of the pulmonary veins with a Teflon patch covering the caval orifices and the mitral valve (Fig 2). The "new" pulmonary atrium was enlarged with a heterologous pericardial patch. The cardiopulmonary bypass was discontinued without difficulty with the patient in sinus rhythm. Pressures of 40 mm Hg in the left (pulmonary) ventricle, 80 mm Hg in the right (systemic) ventricle, and 25 mm Hg in the right pulmonary artery were measured. The hemodynamics produced by the muscular ventricular septal defects were judged acceptable and therefore pulmonary banding was not considered.

After a short period with signs of low cardiac output requiring mechanical ventilation and inotropic support, the hemodynamic condition gradually improved and the patient was extubated on postoperative day 3. Inotropic support was discontinued on postoperative day 7.

On day 9 the patient left the intensive care unit. On day 12 an episode of subpulmonary edema requiring tracheal intubation occurred.

With suspicion of excessive pulmonary blood flow a control cardiac catheterization was performed. The measured data were: pulmonary artery pressure, 20/14 to 10 mm Hg; left ventricular pressure, 75/0 to 4 mm Hg; and radial artery pressure, 125/76 to 65 mm Hg with a calculated Q_P/Q_S of 2:1. Based on these data a conservative treatment was decided on and the patient was maintained on continued positive airway pressure for 15 days until she was finally extubated. On postoperative day 40 the child was discharged from the hospital in good condition. A second control catheterization performed 20 months after the operation showed the persistence of only a small apical ventricular septal defect with a pulmonary artery pressure of 20 mm Hg and a Q_P/Q_S of 1.1:1. She is now growing normally and receiving digoxin and diuretics.

Comment

Transposition of the great arteries combined with TAPVC is a very rare congenital heart disease. When the two

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Table 1. Catheterization Data

Sampling Site	Oxygen Saturation (%)	Pressure (mm Hg)
Superior vena cava	60	
Right atrium	86	3 (mean)
Inferior vena cava	71	
Right ventricle	84	80/0 to 2
Pulmonary artery	84	35/25 to 15
Pulmonary veins	96	
Left atrium	83	2 (mean)
Left ventricle	84	70/0 to 3
Ascending aorta	85	80/60 to 40

congenital heart defects occur simultaneously in the same patient, one defect partly corrects the negative hemodynamic consequences of the other. Total anomalous pulmonary venous connection delivering all pulmonary venous return to the systemic side of the heart avoids overloading of the pulmonary circulation, different from the situation with the torrential pulmonary blood flow occurring when TAPVC is an isolated anomaly.

Probably TAPVC with TGA presents the best flow pattern for an adequate mixing between the two circulations. In fact our patient had complete mixing, and this fact may explain the survival until 16 years of age of the patient with simple TGA and the supracardiac type of TAPVC described by Sapsford and associates [3]. Our patient (despite the gradient across the left ventricular outflow tract) had an increased total pulmonary blood flow requiring operation in infancy.

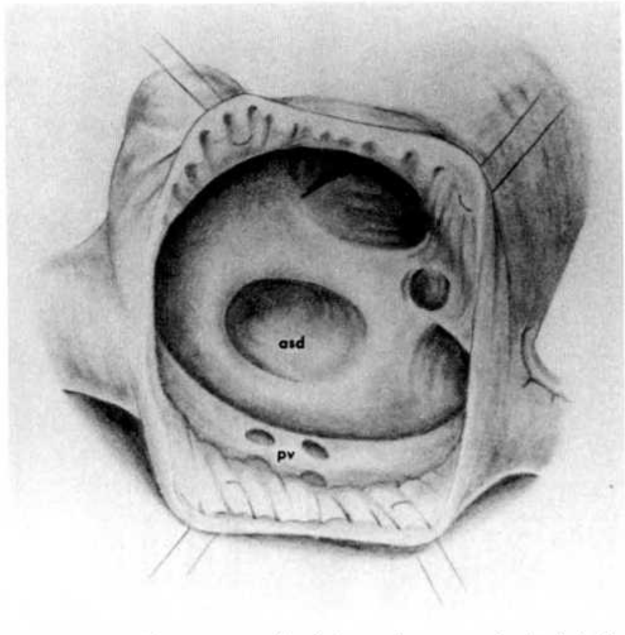

Fig 1. Intraatrial anatomy, with all four pulmonary veins (pv) *draining into the right atrium.* (asd = *atrial septal defect.)*

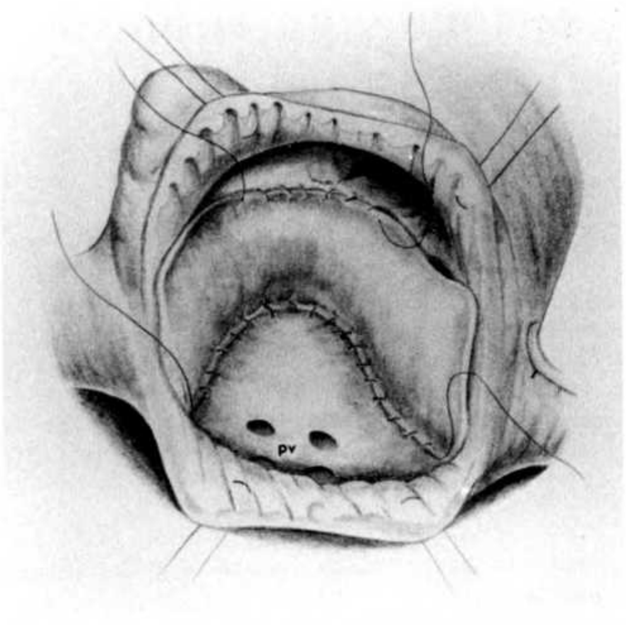

Fig 2. Tunnel rerouting of the caval blood to the mitral valve (modified Mustard operation). (pv = *pulmonary veins.)*

In the postoperative period, even if the Q_P/Q_S remained almost identical, the total blood volume handled by the heart was substantially reduced, with a reduction of the total pulmonary blood flow per se. In this new hemodynamic pattern, the presence of the multiple ventricular septal defects could be well tolerated.

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Norwood Operation for Univentricular Heart With Subaortic Stenosis in the Neonate

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In the setting of a single ventricle, subaortic stenosis may be enhanced by pulmonary artery banding and may later contraindicate a Fontan operation. The Norwood operation may prove a preferable alternative in some infants as a preparatory procedure. We have successfully used this procedure as the initial operation to palliate a newborn with tricuspid atresia, transposition of the great arteries, coarctation, and severe arch hypoplasia secondary to a restrictive bulboventricular foramen.

(Ann Thorac Surg 1990;50:822–5)

Spectacular advances have been made in the surgical treatment of hypoplastic left heart syndrome. What used to be a virtually uniformly fatal condition can now be successfully palliated in the newborn period [1]. We report on the successful application of the Norwood operation in a patient with tricuspid atresia, transposition of the great arteries, coarctation, and severe arch hypoplasia resulting from a restrictive bulboventricular foramen.

A 2.2-kg baby boy was transferred to The Montreal Children's Hospital on the seventh day of life for respiratory distress and congestive heart failure. Echocardiography revealed the diagnosis of (S,D,D) tricuspid atresia, transposition of the great arteries, coarctation, and marked hypoplasia of the aortic arch (Fig 1). Decreasing systemic perfusion was reversed by the administration of prostaglandins to maintain ductal patency. Balloon atrial septostomy performed at cardiac catheterization confirmed the diagnosis and demonstrated a restrictive bulboventricular foramen leading into the subaortic chamber (Fig 2).

Operation was performed at 10 days of age. The following observations were made: the ascending aorta was 6 mm in diameter; the aortic arch between the innominate artery and the left common carotid artery was 3 mm in diameter, tapering to 2 mm distal to it. The main pulmonary artery was 14 mm in diameter and the right and left pulmonary arteries were well developed (Fig 3). The baby underwent the Norwood operation (Fig 4). Under deep hypothermic circulatory arrest, the main pulmonary artery was transected at its bifurcation and the distal end closed with a patch of aortic homograft. The patent ductus was ligated and divided. The concave surface of the aortic arch was incised and the incision was carried distally 10 mm past the area of coarctation. Proximally the incision was carried into the ascending aorta up to the level of the stump of the main pulmonary artery. The aortic arch was augmented with an appropriately fashioned aortic homograft patch well beyond the area of coarctation. Proximally the reconstruction involved the main pulmonary artery stump, which was sutured to the side of the ascending aorta. Pulmonary artery flow was provided by a 4-mm Gore-Tex shunt from the undersurface of the newly reconstructed aortic arch and the confluence of the right and left pulmonary arteries. The sternum was not closed at the end of the operation because of cardiopulmonary compromise, but was successfully closed 2 days later.

The baby was extubated 8 days postoperatively. He was kept in the hospital 5 weeks after the operation because of prematurity. At 9 months of age, increasing cyanosis was noted and a cardiac catheterization was carried out (Fig 5). The baby underwent a right modified Blalock-Taussig shunt with a 6-mm Gore-Tex graft and take-down of the previous central shunt. Now, at the age of 2 years, a Fontan operation is anticipated.

Comment

There is a group of patients who have the physiological equivalent of hypoplastic left heart syndrome and require complex palliative aortic reconstruction in the newborn period to achieve survival. We are reporting on the case of a patient with tricuspid atresia and transposed great arteries with coarctation and severe aortic arch hypoplasia secondary to a restrictive bulboventricular foramen, which resulted in decreased forward flow to the ascending aorta. In such a patient, a substantial part of the systemic perfusion, especially to the lower body, is provided by the patent ductus arteriosus. It is doubtful that a more classic approach such as coarctation repair and pulmonary artery banding would result in survival. It is possible to carry out a complex reconstruction of the aortic arch via a left thoracotomy as reported by Elliott [2]. However, the restrictive bulboventricular foramen would remain and the application of the pulmonary artery band would further accelerate its reduction in size by stimulat-

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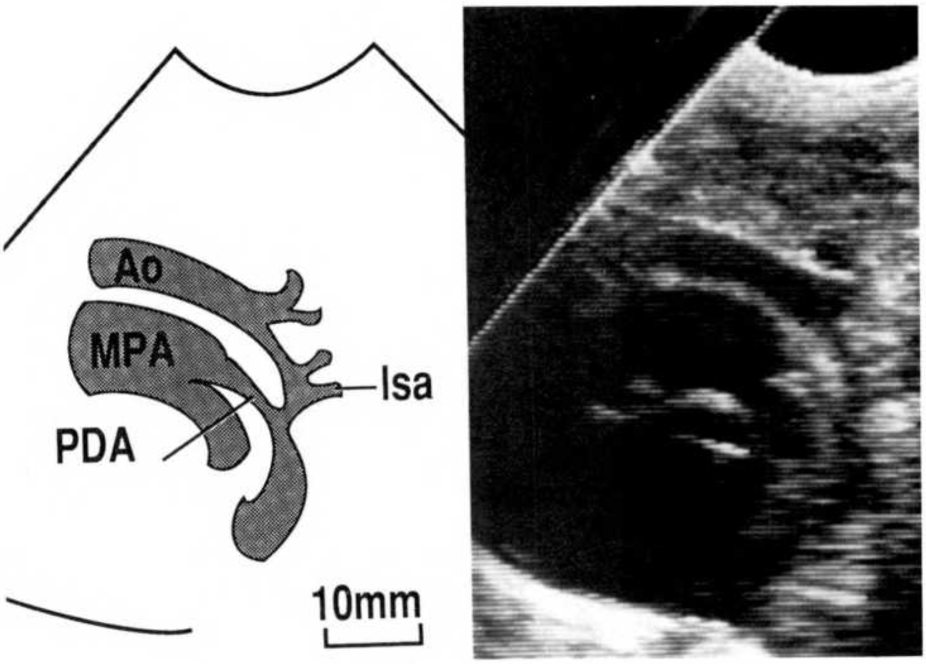

Fig 1. Two-dimensional echocardiography demonstrating the hypoplastic aortic arch. (AO = aorta; lsa = left subclavian artery; MPA = main pulmonary artery; PDA = patent ductus arteriosus.)

ing ventricular hypertrophy. The resultant subaortic stenosis, we have learned, is very difficult to treat and adversely affects survival of these patients [3, 4].

Jonas and colleagues [5] have recently reported 3 patients in whom subaortic stenosis developed rapidly after the application of a pulmonary artery band. All 3 patients were converted to a Norwood circulation between 4 and 10 weeks of age because of the presence of severe subaortic stenosis. It is becoming clear that patients with single ventricle and clinically significant subaortic stenosis have a higher mortality after the Fontan operation [6, 7] or ventricular septation [8]. Furthermore, Freedom and colleagues [9] have clearly identified pulmonary artery band-

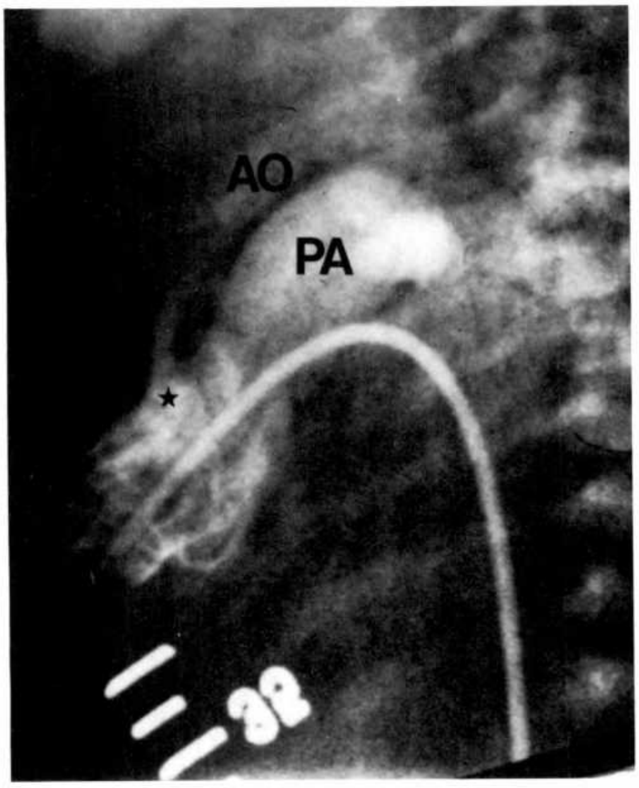

Fig 2. Cardiac catheterization showing the subaortic stenosis (★). The ascending aorta (AO) is much smaller than the main pulmonary artery (PA).

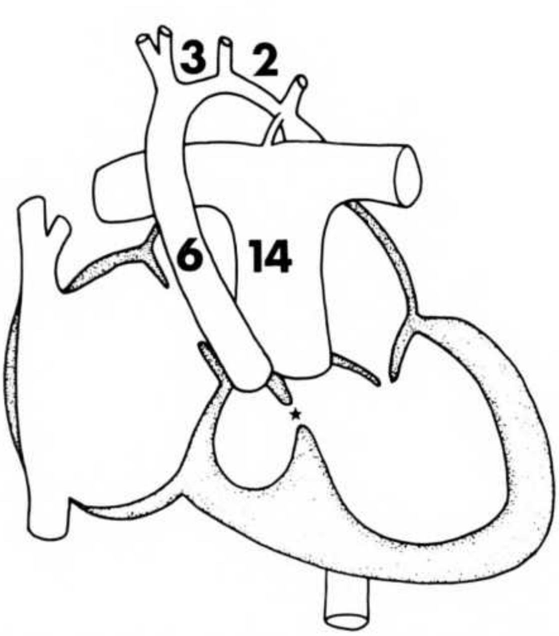

Fig 3. Operative findings of subaortic stenosis (★) and the relative sizes of the great vessels. All numbers indicate diameter in millimeters.

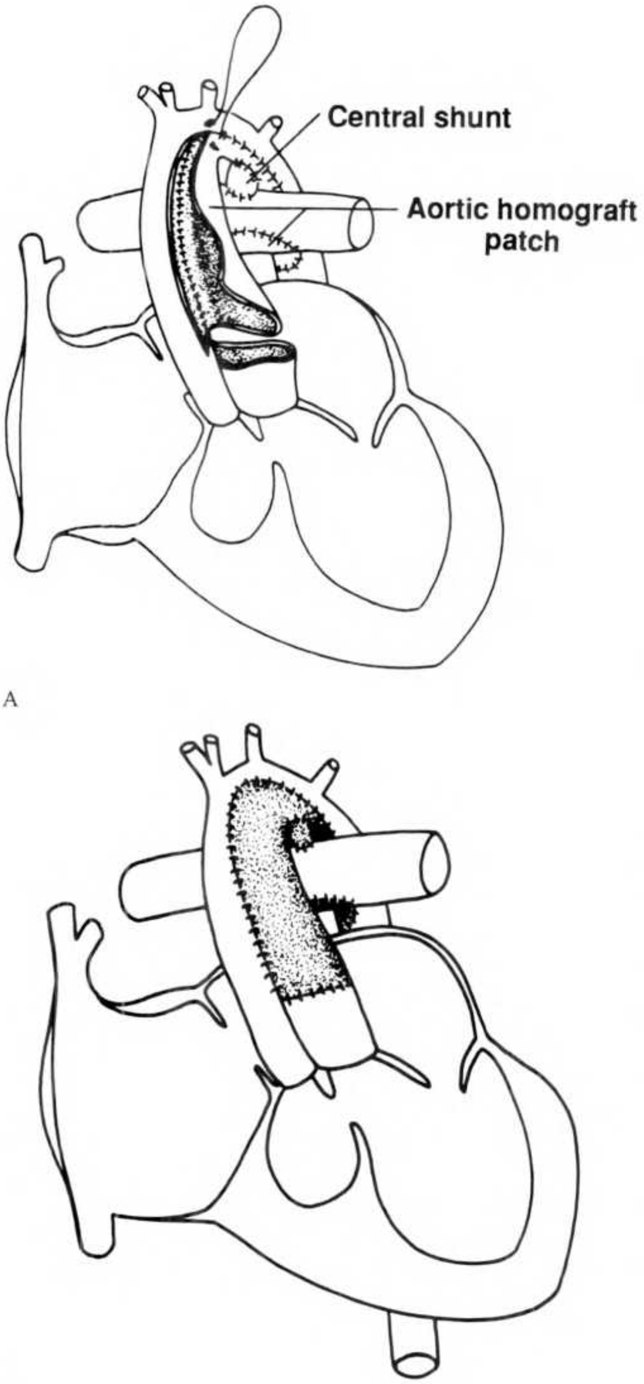

Fig 4. The Norwood operation: (A) An aortic homograft is used to reconstruct the ascending aorta and hypoplastic aortic arch as well as to close the distal main pulmonary artery. (B) The completed reconstruction.

ing as a significant etiological factor in the development of subaortic stenosis in patients with univentricular heart. The pulmonary artery band will stimulate marked myocardial hypertrophy, which will undoubtedly alter ventricular compliance and impair atrial emptying. Kirklin and colleagues [10], in a review of their experience with the Fontan operation, uncovered hypertrophy of the main ventricular chamber as a significant risk factor in both early and late increased mortality.

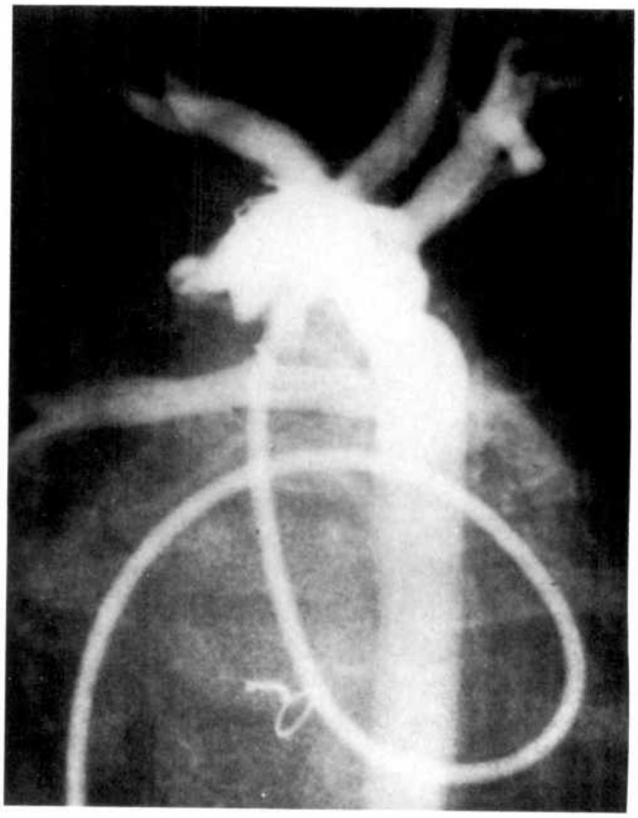

Fig 5. Postoperative angiogram done at 9 months of age showing the reconstructed aortic arch.

We believe that the only hope for long-term survival in these patients is the achievement of unobstructed systemic circulation, both at the egress from the heart and at the aortic arch level, and preservation of myocardial function by avoidance of stimulus for ventricular hypertrophy. By necessity, pulmonary blood flow is to be provided by an aortopulmonary shunt.

The Norwood operation fulfils these goals, and its indication should be expanded so that in addition to patients with hypoplastic left heart syndrome, it would include patients like ours and be performed as the initial primary palliative reconstruction.

In conclusion, we feel that the successful treatment of subaortic stenosis in the univentricular heart begins with its early diagnosis in the neonatal period and recognition of the types of anatomical situations that favor its subsequent development. The Norwood operation is an important addition in the surgical management of these patients.

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REVIEW OF RECENT BOOKS

Surgery of the Chest, 5th Edition, Volume II
Edited by David C. Sabiston, Jr, MD, and
Frank C. Spencer, MD
Philadelphia: W.B. Saunders
2,084 pp, illustrated, $250.00

Reviewed by Anthony R. C. Dobell, MD

Volume II of Sabiston and Spencer's fifth edition of *Surgery of the Chest* is primarily devoted to acquired and congenital cardiac surgery. Each chapter has been prepared by recognized authorities, many of whom have made the original contributions in the area under discussion.

Chapters are written according to a uniform framework. Historical aspects are usually briefly described; pathophysiology and clinical features are succinctly recorded. Surgical management and postoperative care are extensively described and updated. The quality and quantity of figures vary considerably. Postoperative results and long-term management are generally thoroughly described and supported by the most recent literature.

A strong point of this textbook is the selected bibliography that accompanies each chapter; the authors have chosen key articles that are very well summarized. The bibliography itself is always extensive, and this is a major reason for the book's extreme usefulness to cardiothoracic trainees. This textbook offers a complete review of almost every aspect of cardiothoracic surgery, and the authors and editors have done a remarkable service in compressing an extraordinary amount of data into a manageable form.

Montreal, Quebec, Canada

Circumflex Artery Ventricular Fistula and Pseudoaneurysm After Mitral Reoperation

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Prosthetic mitral valve reoperation complicated by atrioventricular groove pseudoaneurysm and circumflex ventricular fistula is presented. Ligation of the circumflex artery during mitral valve replacement is implicated after review of a previous cardiac angiogram.

(Ann Thorac Surg 1990;50:826–7)

Cardiac reoperation after previous mitral valve replacement has many possible complications. Some of these are ventricular rupture, pseudoaneurysm formation [1], paravalvular leak, prosthetic valve malfunction, and damage to the circumflex artery from sutures placed too deeply [2]. This case represents an unusual combination of complications causing a circumflex artery ventricular fistula and pseudoaneurysm.

A 47-year-old woman was seen for routine examination and was found to have a new murmur. This patient had a past history of mitral valve replacement with a No. 31 Beall valve in 1978 for rheumatic mitral valvar disease. She did well until the Beall valve malfunctioned, associated with severe mitral regurgitation and heart failure in 1987. At that time, she had reoperation and a No. 31 St. Jude mitral valve replacement was done with interrupted pledgetted sutures. The procedure as reported was done in a routine fashion; however, the adhesions over the left ventricular posterior wall were not taken down. She was taken from the operating room with low-dose inotropic support and without serious bleeding. The remainder of her hospital stay was uneventful. She did well and only had complaints of occasional palpitations during her routine visits.

On examination 2 years later, a new diastolic murmur was auscultated. An echocardiogram and computed tomographic scan of the chest showed a large mass protruding from the posterior wall of the left ventricle into the left chest. Cardiac catheterization showed a proximal circumflex artery ventricular fistula (Fig 1) and a large pseudoaneurysm (Fig 2) extending from the atrioventricular groove. The prosthetic valve, however, functioned normally and without any leak.

She subsequently was taken to the operating room, where for the third time her left atrium was opened at the right, superior to the pulmonary veins. Her St. Jude valve was removed. A large pseudoaneurysm was seen in the atrioventricular groove extending around approximately 50% of the annular circumference from the 5-o'clock to the 11-o'clock position. The neck of this aneurysm was approximately 3 cm. A Dacron patch was then sewn over the opening into the pseudoaneurysm. The circumflex ventricular fistula was ligated. A No. 31 St. Jude mitral valve was re-replaced into the mitral annular area, and the patient was taken from the operating room uneventfully. The only complication occurring postoperatively was reoperation for bleeding, which was done uneventfully.

Comment

Although left ventricular pseudoaneurysm is occasionally reported after mitral valve replacement [3], in this case we report circumflex ventricular fistula after redo mitral valve replacement. There are a few explanations for these complications, but most likely they were caused by infarction [4] after inadvertent suturing of the circumflex artery while replacing her valve the second time. Circumflex artery ligation at the time of mitral valve replacement usually results in operative death, but in those who survive, chronic aneurysm formation is frequently seen. MacVaugh and associates [5] reported postmortem examinations on 4 similar cases, showing the circumflex artery to be ligated 2.5 to 3.5 cm from its origin. This site corresponds to where the artery is closest to the mitral annulus. In our patient a large mitral valve was initially placed, possibly compressing the mitral annular tissue toward the circumflex artery, as seen in her preoperative angiogram (Fig 3). It is easily seen in the figure how the circumflex artery could be suture-ligated after prosthetic valve excision and replacement. The ensuing myocardial infarction was relatively unnoticed in the initial postoperative period. Subsequent myocardial necrosis or hematoma formation evolved to left ventricular rupture and fistula. The rupture was well contained within the myocardium because the left ventricular pericardial adhesions were not taken down at the time of her second mitral valve replacement. No symptoms developed in our patient, but coronary artery fistulas may cause ischemia-related steal or siphon effect [6], endocarditis, arterial aneurysm formation, and congestive heart failure related to increased ventricular volume similar to that of aortic insufficiency. Operation is similarly indicated for left ventricular pseudoaneurysms [7], which can cause symptoms from extrinsic pressure and rupture.

We decided the best surgical approach would be the same as that for acute left ventricular rupture associated

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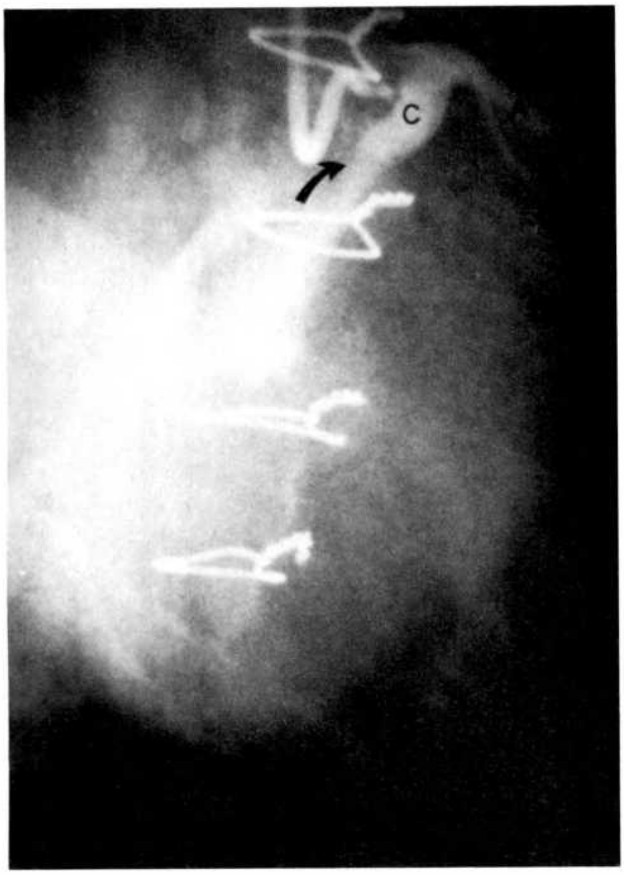

Fig 1. Cardiac angiogram showing an aneurysmal proximal circumflex coronary artery with fistula to the left ventricle. (A = *anterior descending coronary artery;* C = *circumflex artery.)*

with mitral valve replacement. We therefore removed the perfectly functioning mitral valve, ligated the coronary fistula through the defect, patched the defect with Dacron to limit tension on the residual myocardium, and then re-replaced the valve. We believe that this was clearly better than going through the pseudoaneurysm with expectations of being able to sew one side of the patch to the mitral prosthesis. With our method the visualization was excellent, and we believe that this was a safer, although more time-consuming, approach. Finally, there is an increased propensity toward these complications with any mitral prosthetic reoperation. We believe the safe removal of the mitral valve requires cutting of the old sutures and extraction of the valve, thereby preserving the maximum amount of annulus for resuturing. The pitfalls of mitral valve replacement and the circumflex anatomy are always present and are vividly seen in our presentation.

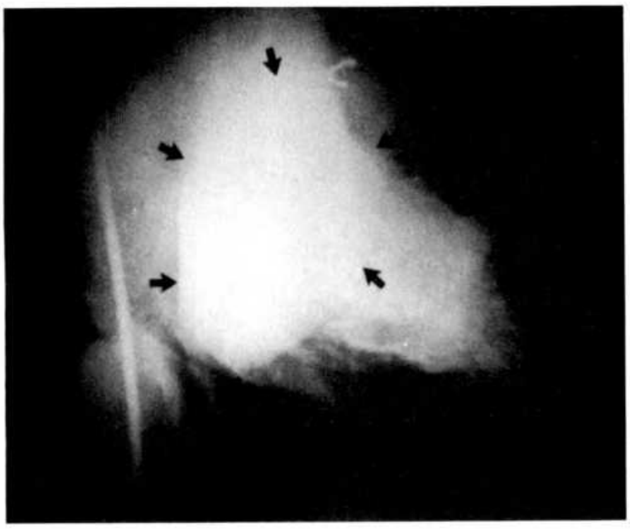

Fig 2. Right anterior oblique projection of ventriculogram with arrows *demonstrating double density of atrioventricular groove pseudoaneurysm.*

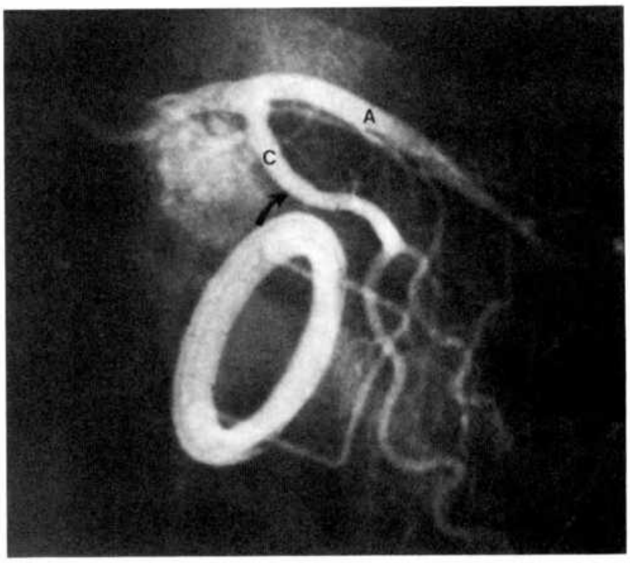

Fig 3. Cardiac angiogram after first mitral valve replacement showing the proximity of the circumflex artery to the mitral annulus. An arrow *indicates subsequent site of injury.* (A = *anterior descending coronary artery;* C = *circumflex coronary artery.)*

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Mediastinal Hibernoma, A Rare Tumor

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Hibernoma is an uncommon soft tissue tumor that is derived from the remnants of fetal brown fat. Review of the world medical literature revealed 90 cases, 6 of which were intrathoracic. We present the seventh case of intrathoracic hibernoma; in this case, the hibernoma was within the mediastinum without direct invasion of other structures.

(Ann Thorac Surg 1990;50:828–30)

Hibernoma is a rare, benign soft tissue tumor composed of multiloculated fat cells derived from brown fat. The tumor was first described by Merkl [1] in 1906 as being composed of brown adipose tissue. The term *hibernoma* was proposed in 1914 by Gery [2] because of its morphologic similarity to the cells of the so-called hibernating gland of animals.

By 1985, 80 cases of hibernoma were reported in the world medical literature [3]. The total grew to 89 in 1988 [4], and with an additional case reported in 1989 [5], the current total stands at 90. Of six reported intrathoracic hibernomas, five were confined within the subpleural chest [6–10], and one was intramyocardial [11].

A 16-year-old Filipino boy was referred to the Thoracic Surgery service at Kaiser Permanente Medical Center, Los Angeles, CA, on January 20, 1989, because of a large anterior mediastinal mass that was discovered on a routine preemployment physical examination. The patient was in excellent health and completely asymptomatic. The past medical history and physical examination were noncontributory.

A chest roentgenogram demonstrated a large mass in the left anterior mediastinum, inseparable from the cardiac silhouette (Fig 1). Computed tomography showed a 8 × 8 × 12-cm mixed-density mass, which was mainly fatty, in the left mediastinum, abutting the aortic and pulmonary arches and the left side of the heart without obvious invasion (Fig 2).

On February 1, 1989, a median sternotomy was performed. A large, tan, lobulated and encapsulated, bilobed mass was found in the anterior-superior mediastinum. It was completely within the mediastinal pleura and extrapleurally compressed the upper segment of the left lung. The left phrenic nerve was easily dissected free of the mass. The pericardium and major vascular structures were not involved. The tumor was highly vascular but was removed without complication. The postoperative course was uneventful. The patient was discharged on the third postoperative day and has not shown any evidence of recurrence after 1 year.

The fresh specimen was tan-brown, homogeneous, and encapsulated, with a buttery consistency. Attached to the specimen was a portion of a normal-appearing thymus that weighed 42 g and measured 8 × 3 × 3 cm.

Light microscopic examination showed tumor cells arranged in lobules separated by fine reticulin fibers. Most of the cells were round or polygonal with monovacuolated or multivacuolated lipid-containing cytoplasm (Fig 3). These were mixed with cells having a less vacuolated, eosinophilic, granular cytoplasm. The nuclei were ovoid and spherical and central. A delicate fibrocollagenous and vascular capsule was present with inconspicuous mesothelial cells on the surface. The sections of thymus were normal.

Comment

Hibernomas are rare, benign tumors derived from brown adipose tissue, a specialized form of fat found in hibernating and nonhibernating animals. Brown fat was first

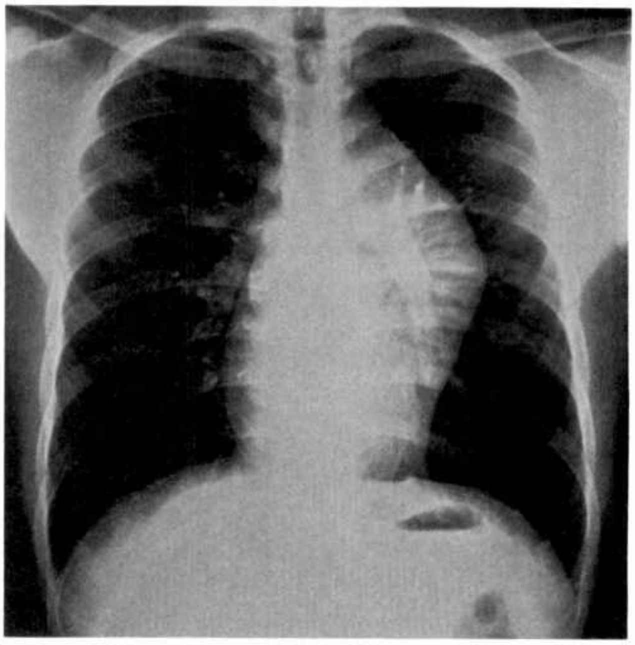

Fig 1. Chest roentgenogram showing large opacity in left anterior mediastinum.

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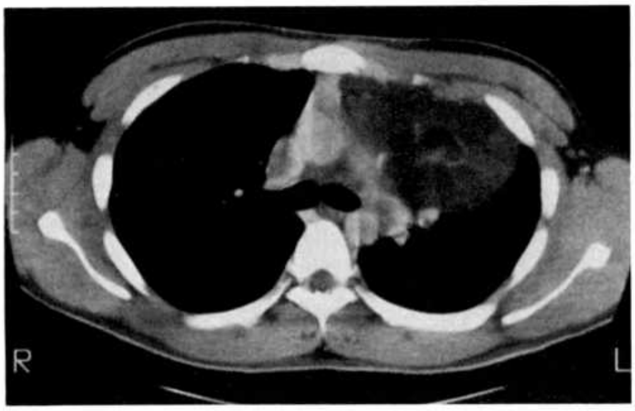

Fig 2. Computed tomographic scan shows that the mass has a mixed density but is mainly fatty.

described by Velch in 1670. He noted a glandlike structure in the mediastinum of a woodchuck, a hibernating animal, and thought it to be associated with the thymus [12]. This was later recognized by Barkow in 1846 to be separate from the thymus, and he called it the "hibernating gland" [12]. Rasmussen [12] reviewed the morphology of this structure and discovered its presence in more than 50 species including nonhibernating animals such as rats, rabbits, cats, monkeys, and humans.

In 1902, Shaw [13] demonstrated the axillary and subpleural fat of the human fetus and newborn to be largely brown fat, in contrast to the yellow fat of subcutaneous tissues. Finally, Bonnot [14] reported that brown fat in humans persisted through the embryonic, neonatal, and adult stages in those locations described in the embryo and the fetus.

In the adult, brown fat is usually found in scattered foci along the esophagus, trachea, large vessels of the mediastinum, posterior neck, and interscapular regions. Therefore, it is not surprising that the distribution of these tumors closely follows the sites of persistence of brown fat in the human adult as shown by the previously mentioned anatomical studies. The most common site of reported hibernomas is the subcutaneous tissues of the back, especially the interscapular area. Other frequent locations include the neck, axilla, thigh, and intrathoracic area [15].

A review of the world literature brings the total number of reported cases of hibernoma to 90. There have been 5 previously reported cases of intrathoracic hibernoma, all of which were confined to the subpleural chest and did not invade the mediastinum. All of these patients underwent successful thoracotomy and resection of tumor. None had evidence of recurrence after 1 year (Table 1). There has been 1 reported case of intramyocardial hibernoma, as revealed by an autopsy study of an 84-year-old woman [11]. The case presented here is the only other known reported case of mediastinal hibernoma.

Although these tumors are considered benign and malignant transformation has not been reported, they tend to grow to large proportions ranging in size from 3 to 19 cm [15]. Because hibernomas are well encapsulated and do not show infiltrative growth, they can be totally removed without sacrificing vital structures. Therefore, in the treatment of these tumors, especially if intrathoracic in location, surgical excision is curative.

With the increasing incidence of routine chest roentgenograms, most of these tumors will be detected as asymptomatic opacities, as in the case presented here. These tumors tend to grow to large proportions and will eventually produce signs and symptoms accordingly, as noted in previous reports. For this reason and on the basis of their clinically indeterminate nature, complete surgical excision should be performed.

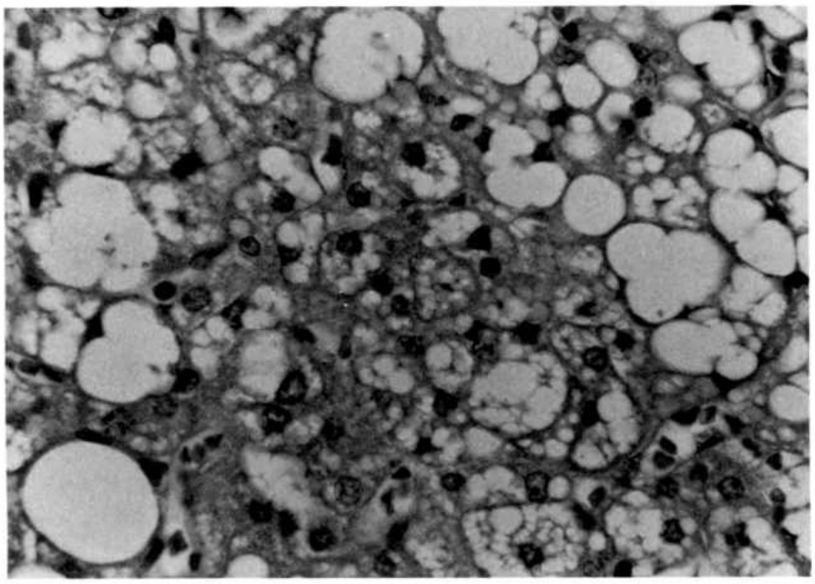

Fig 3. Cells with multivacuolated cytoplasm mixed with cells having a less vacuolated, eosinophilic granular cytoplasm. (Hematoxylin and eosin, ×400 before 38% reduction.)

Table 1. Clinical Features of Reported Intrathoracic Hibernomas

Author	Age (y)	Race	Sex	Signs/Symptoms	Tumor Location	Tumor Size (cm)
Kittle et al [6]	42	W	F	Cough, wheeze	Posterior base, left chest	14 × 12 × 8
Gross and Wood [7]	39	W	M	Left pleuritic pain, left arm paresthesias	Apex, left chest	9 × 8 × 4
Peabody et al [8]	31	B	M	Left pleuritic pain	Apex, left chest	3 × 3 × 2
Beetstra and Quast [9]	22	W	M	Asymptomatic	Apex, right chest	5 × 4 × 3
May et al [10]	26	W	M	Asymptomatic	Middle posterior, left chest	4 × 4 × 4
Kindblom [11]	84	W	F	Sinus arrhythmia, LBBB	Right atrium	4 × 4 × 3

B = black; LBBB = left bundle-branch block; W = white.

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Acute Hypercalcemic Crisis After an Open Heart Operation

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Acute hyperparathyroidism developed in a previously normocalcemic 64-year-old woman during the first week after a coronary operation. Prolonged QT interval in the electrocardiogram and hypercalcemia were documented on the fourth postoperative day. Neck exploration on the fifth postoperative day revealed a lower right parathyroid adenoma. Parathyroidectomy resulted in rapid and dramatic improvement of the clinical picture and normalization of laboratory values.

(Ann Thorac Surg 1990;50:831–2)

Acute hypercalcemic crisis is the most dramatic form of symptomatic hyperthyroidism [1]. Acute hypercalcemic crisis developed after a coronary bypass operation in 1 of our patients without preoperative clinical evidence of hyperparathyroidism.

A 64-year-old woman was admitted to the cardiothoracic unit for coronary artery bypass grafting. Angina pectoris had developed 6 months before her admission, and she was subsequently found to have stenoses of the three major coronary arteries. The medical history revealed chronic polyarthritis being treated with steroids, stable adult diabetes mellitus well compensated by oral medication, and one episode of renal colic without evidence of stones 10 years earlier.

Preoperative blood chemistry and hematological values were within normal limits. The electrocardiogram did not show any abnormality. Two days after admission the patient underwent triple aortocoronary bypass grafting. During the operation intravenous steroid substitution was started. In the intensive care unit she had an uneventful course and was extubated the morning after operation. The patient, at the time fully oriented and hemodynamically stable, was able to be transferred to the ward.

On postoperative day 2 the patient complained of tiredness and of nausea, which became worse the next day, when she vomited several times. The initially slight tiredness slowly turned into lethargy by the end of the fourth day. Meanwhile the laboratory variables showed an increase of serum creatinine. Adequate urine output could be maintained only with crystalloid infusions and administration of diuretics. Blood glucose level was normal.

On the fifth postoperative day the electrocardiogram showed a shortening of the QT interval to 0.24 ms; this finding was highly suggestive of hypercalcemia. The serum calcium level was indeed elevated (3.45 mmol/L [13.84 mg/dL]), and the parathyroid hormone level was very high (1,145 ng/L; upper normal limit of mid-C-regional 44–68 parathyroid hormone level, measured by radioimmunoassay, is 300 ng/L) [2, 3].

With the diagnosis of acute hyperparathyroidism most likely due to a parathyroid adenoma the patient underwent neck exploration. A 2 × 3-cm lower right parathyroid adenoma was found along with three other normal glands and resected. The diagnosis of adenoma was confirmed histologically.

After the operation serum calcium level decreased rapidly (Fig 1) and the patient's condition improved within a couple of hours. Her state of consciousness was normal 12 hours after parathyroidectomy. The further postoperative course was uneventful and the patient was discharged home a week later. Presently, 1 year after operation the patient is normocalcemic and free from angina with a negative exercise test.

Comment

Acute hyperparathyroidism in a previously normocalcemic patient is a rare occurrence. The rarity of the condition and the lack of pathognomonic symptoms make its recognition particularly difficult [4].

In our patient the preoperative laboratory values were all within normal limits and she had no clinical evidence of long-standing hypercalcemia, although she must have had the adenoma for some time. It could be conceivable that a slow but steady increase in parathyroid hormone level may have activated renal and osseous compensatory mechanisms that kept the disease subclinical. The long-standing steroid therapy also might have played a role by increasing renal elimination of calcium, although its beneficial role in primary hyperparathyroidism remains controversial [5]. Multiple metabolic and endocrine alterations take place during cardiopulmonary bypass [6], and organ hypoperfusion may occur. The triggering event of the hyperparathyroid crisis in such a wide spectrum of homeostatic changes is difficult if not impossible to identify.

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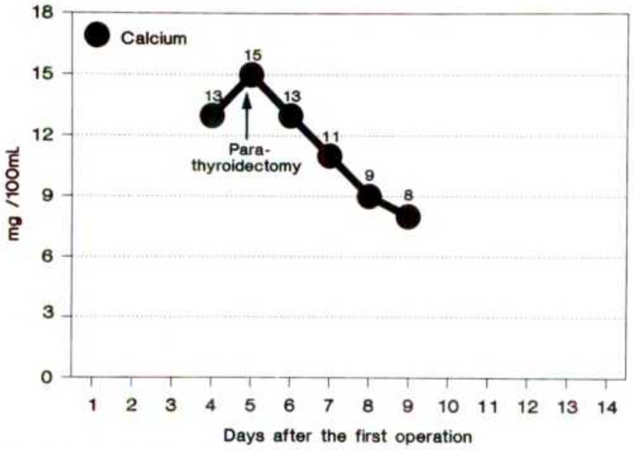

Fig 1. Serum calcium values after open heart operation.

Rapid and short-lasting changes in serum calcium levels may also occur during or shortly after cardiopulmonary bypass, generally caused by the administration of calcium gluconate as an inotropic agent. Our patient received 1 g of calcium gluconate during weaning from cardiopulmonary bypass. This temporary increase in serum calcium level may also have been responsible for the ultimate derangement of calcium metabolism.

Whatever the pathogenetic mechanism might have been, we recognized the clinical manifestation of acute hyperparathyroidism only after 4 days when the patient was already comatose. Fortunately, shortening of the QT interval on the electrocardiogram was noted, which gave us the clue for the correct diagnosis. Electrocardiographic changes during hypercalcemia are by no means a consistent finding; they occur in only half of the patients with the syndrome [4]. Routine postoperative serum calcium level determination would have surely speeded up the diagnosis but, as this case is rather exceptional, one would question the necessity of including serial serum calcium determinations in the preoperative and postoperative laboratory routine.

Patients whose condition is deteriorating under vigorous medical treatment and those who are already lethargic or comatose at the time of diagnosis should primarily undergo surgical exploration of the neck to remove the adenomatous or hyperplastic parathyroid tissue. The results of this aggressive approach are often rewarding, with long-term normocalcemia and a drastic reduction in mortality [7].

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Easy Cardioplegia Delivery, Aortic Venting, and Reperfusion Technique

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Myocardial preservation is an essential aspect of coronary revascularization as well as of other open heart procedures. As these procedures are becoming more complex, specifically myocardial revascularization, impeccable myocardial protection becomes imperative. Described is a technique that enables one to easily accomplish good myocardial preservation with minimal clutter using readily available materials.

(Ann Thorac Surg 1990;50:833–5)

Myocardial preservation and techniques to prevent reperfusion injury are critical to successful open heart operations. Many innovative techniques have been detailed in the literature [1–6]. Described herein is a technique that allows easy delivery of blood or crystalloid cardioplegia, warm or cold, antegrade or retrograde. It can be accomplished with readily available manufactured delivery sets or with generic versions constructed in the operating suite. This technique has been employed by me in 463 open heart surgical cases in an 18-month period with no related complications. The incidence of postsurgical cerebrovascular events in these patients has been less than the overall occurrence of postsurgical cerebrovascular events in patients undergoing similar procedures at Emory University Hospital. There have been no instances of aortic dissection. In the past year, in approximately 50% of the open heart surgical procedures performed at Emory University Hospital this technique or a similar version has been used.

Technique

Cardiopulmonary bypass is instituted in the standard fashion. A pursestring of 4-0 polypropylene suture is placed in the ascending aorta at a proposed proximal vein graft site. A 12-gauge aortic root vent Y cardioplegia cannula (No. 20012 aortic root cannula with vent line; DLP, Inc, Grand Rapids, MI) is placed within the pursestring, which is then snared and secured to the cannula. The 12-gauge cannula is used to minimize the pressure gradient across the system. The gradient measured from the cardioplegia reservoir to the aorta ranges from 125 to 150 mm Hg for this system. A multiple perfusion set (DLP No. 14000 multiple perfusion set) is then attached to the aortic root cannula. The vent port is attached to pump suction, after delivery of cardioplegia, for use as an aortic root vent. Air is removed from the multiple perfusion set first by allowing blood from the aortic limb to flush the system. This limb is then occluded and the cardioplegia line is attached and allowed to flush the vein graft limbs, then these are occluded.

Once the distal saphenous vein graft anastomosis is complete, cardioplegia is delivered through the vein graft using a limb of the multiple perfusion set. If multiple

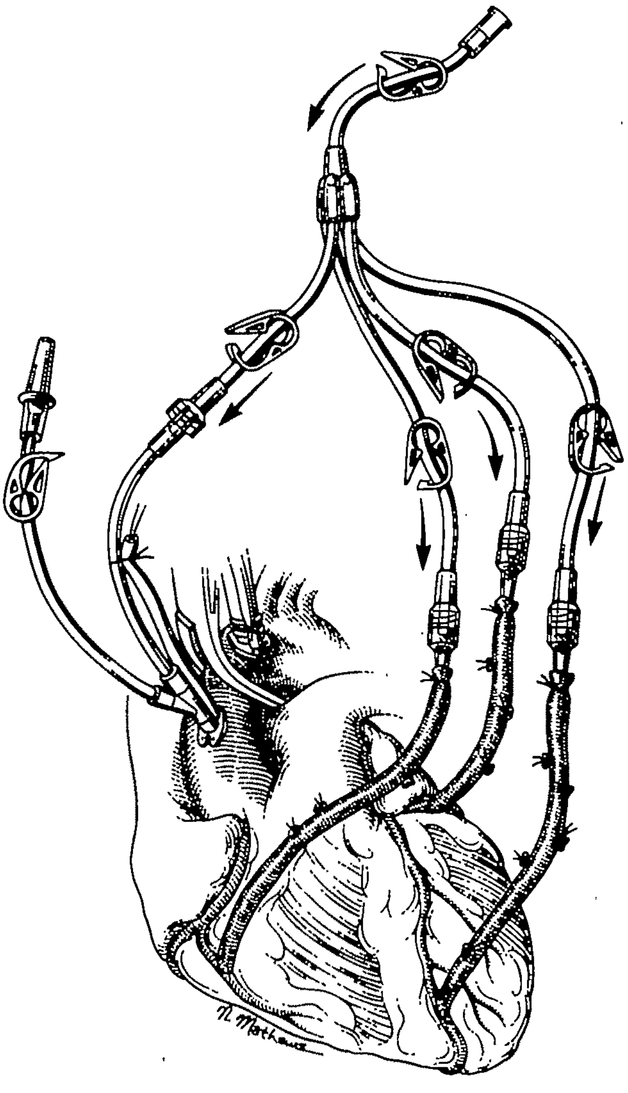

Fig 1. Technique of delivery of cardioplegia into the grafts and aortic root after completion of grafts.

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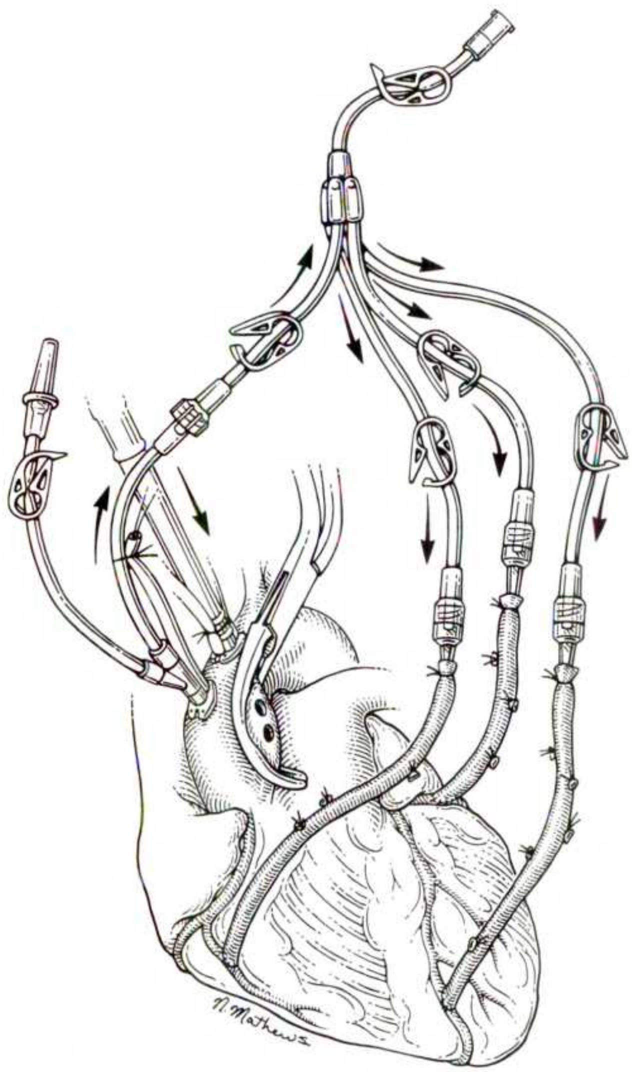

Fig 2. Graft reperfusion after removal of the aortic cross-clamp using the aortic root cannula.

injections of the aortic root are desired, the aortic root cannula is opened and the vent port occluded after cardioplegia has been started, to remove any air in the system. Cardioplegia can be delivered into the aortic root and grafts simultaneously (Fig 1). Pressure of delivery is controlled by the perfusionist using an in-line pressure gauge (DLP pressure display No. 6000); however, the pressure drop across the system must be taken into consideration. In general, we perfuse at a pressure of 200 to 250 mm Hg at the pump with a subsequent aortic root pressure of 100 to 150 mm Hg for the initial injection of antegrade aortic cardioplegia. To be more accurate, pressure in the root can be monitored directly using an aortic root cannula with pressure monitor tip (DLP No. 24009 or equivalent). Once all the grafts are completed, warm blood cardioplegia can be delivered, if desired, to the grafts and the aortic root simultaneously before removal of the cross-clamp [4, 5]. This is usually delivered at a pressure of 50 to 60 mm Hg, measured at the aortic root or graft [1].

The use of retrograde cardioplegia is instituted easily, delivering cardioplegia to the retroplegia cannula and simultaneously applying suction to the aortic root vent. Cardioplegia is not delivered to the vein grafts during the infusion of retrograde cardioplegia. This technique is particularly useful when internal mammary artery grafts are used.

Reperfusion through the grafts after release of the cross-clamp can be accomplished in several ways. One technique is to occlude the suction port. This allows blood to flow from the aorta through the root cannula and out the multiinfusion port into the grafts (Fig 2). Care is taken to remove air from these lines before allowing blood to enter the grafts. Proximal anastomoses are then performed first to the side of the aorta opposite the location of the aortic root cannula. Disadvantages of this method are many: the clamp must be applied to the aorta twice, it can be cumbersome in patients with a small aorta, and the flow to the grafts cannot be monitored. Another method is to remove the aortic root cannula and attach the multiinfusion set to a side port in the aortic cannula (Fig 3)

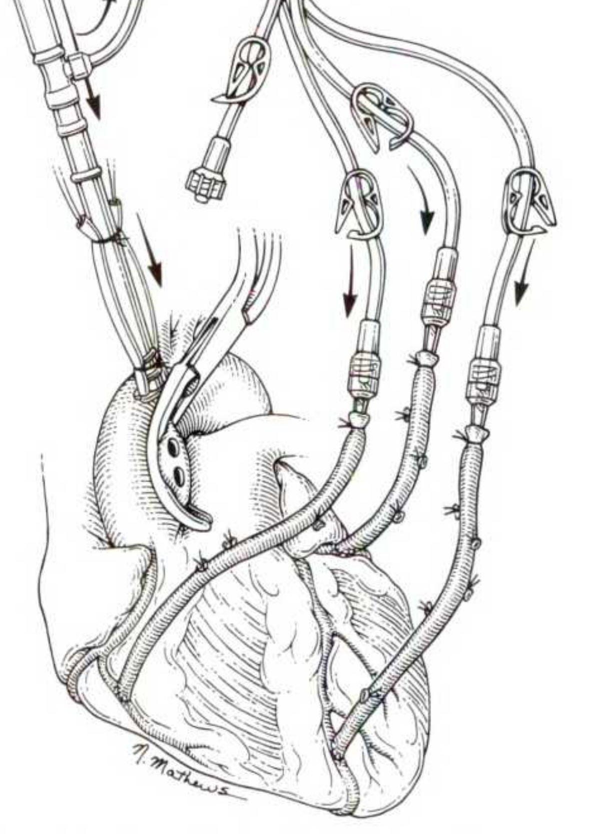

Fig 3. Graft reperfusion after removal of the aortic cross-clamp using the aortic cannula side port.

or to a "bleed line" from the oxygenator providing oxygenated blood. Here again flow cannot be monitored. Our preferred technique is to transfer warm pump blood into the cardiotomy reservoir used for cardioplegia delivery. The perfusionist then uses the same technique of delivery as for cardioplegia, monitoring both pressure and flow.

Comment

This technique of cardioplegia delivery, aortic venting, and reperfusion is easily instituted with minimal clutter. Aortic root suction generally provides a dry, bloodless field during the performance of distal anastomoses and also decompresses the left ventricle. Cardioplegia can be delivered to the grafts or the aortic root during the time of cross-clamping. Retrograde cardioplegia is easily instituted. Terminal injection of warm blood cardioplegia can be delivered to both the aortic root and grafts if desired. Reperfusion through the vein grafts after removal of the cross-clamp is accomplished by one of the methods described. This technique uses products readily obtainable. It allows the surgeon to provide excellent myocardial protection for most cardiac procedures with minimal clutter and without an increase in complications.

Disadvantages of this technique are few. Stroke rate in the 463 patients was 1.1% compared with the 1987 stroke rate of 3.1% at Emory University Hospital with no difference in mortality. There have been no aortic complications due to the cannula. During infusion of cardioplegia into both the aortic root and grafts, a pressure differential can occur. Use of the aortic root cannula with pressure line will at least assure an adequate aortic root perfusion pressure.

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Mitral Prosthetic Replacement in Small Left Atria

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A modified transseptal approach for exposure of the difficult mitral valve is described. This allows the surgeon to visualize the valve, especially during prosthetic replacement, in the presence of a small, friable, or noncompliant left atrium.

(Ann Thorac Surg 1990;50:836–7)

More than 30 years have elapsed since the idea of approaching the mitral valve via the right atrium and the interatrial septum was put into practice [1, 2]. In 1966, Dubost and associates [3] described a new technique based on the same principles that have remained unchanged for the past 24 years, the essence of which is an incision from the right superior pulmonary vein toward the right atrial wall and through the interatrial septum. With the present increasing number of mitral prosthetic replacements, especially in elderly patients, most surgeons have encountered instances wherein explantation of the old and implantation of a new prosthesis has been difficult because of association with a small atrium and friable tissues. The friable nature of the elderly left atrial wall imposes great restraint on the amount of retraction applied if tearing is to be avoided. To reach a compromise for achieving adequate exposure can be, at times, very difficult. Furthermore, because the approach to the left atrium will be through the previous atriotomy suture line, the area is less compliant and tearing occurs more easily. Recently, we have adopted for prosthetic replacement in 29 elderly patients a transseptal technique that differs from the one previously described. This allowed us to approach the mitral valve almost without any traction on the atrial wall and yet achieve optimum exposure. The following technique can be applied in any case of mitral prosthetic replacement as well as first-time mitral valve replacement, if associated with a small atrium. No complications, including atrial arrhythmias, were encountered.

Technique

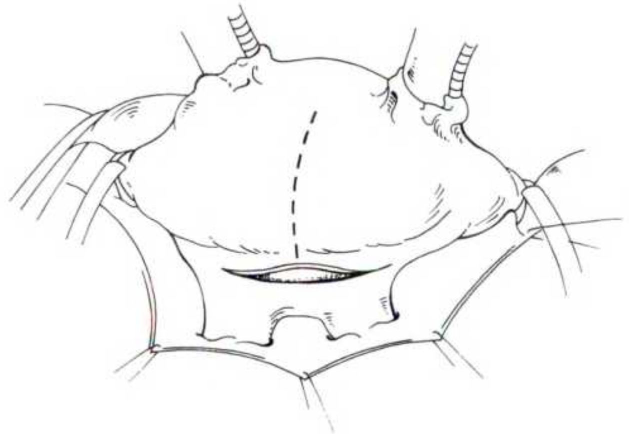

Fig 1. The standard incision in front of the right superior pulmonary vein to approach the mitral valve is shown. If this incision proves inadequate, a vertical one is made between the two caval cannulas on the right atrial wall.

The patient is placed on standard cardiopulmonary bypass. After cardioplegic arrest, both venae cavae are snared and the left atrium is opened with a longitudinal incision in front of the right superior pulmonary vein. A careful attempt is made to achieve optimal visualization of the mitral valve by retracting the superior lip of the atriotomy and, if exposure is considered insufficient or demands too much traction, a vertical incision is made into the right atrium between the two caval cannulas (Fig 1). The interatrial groove is divided and the incision continued anteriorly and obliquely toward the tricuspid annulus, but away from the atrioventricular node area (Fig 2). Retractors are then placed inside the left atrium and, by the application of gentle traction, the mitral valve area is optimally exposed (Fig 3). At completion of the prosthetic replacement the incision in the atrial septum is secured with a continuous monofilament suture including

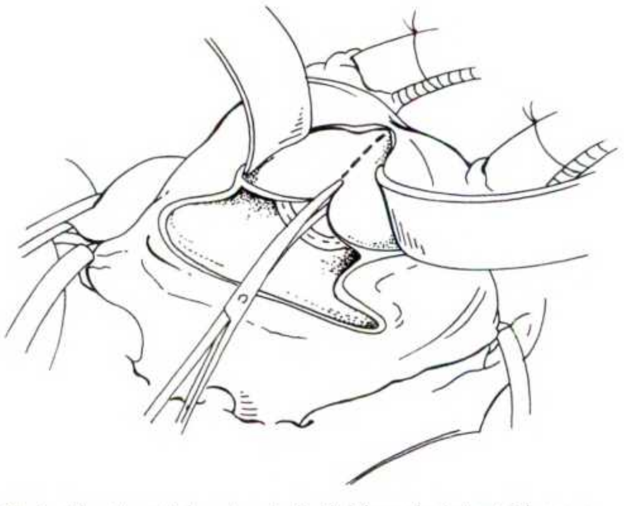

Fig 2. The interatrial septum is divided from the interatrial groove toward the tricuspid annulus.

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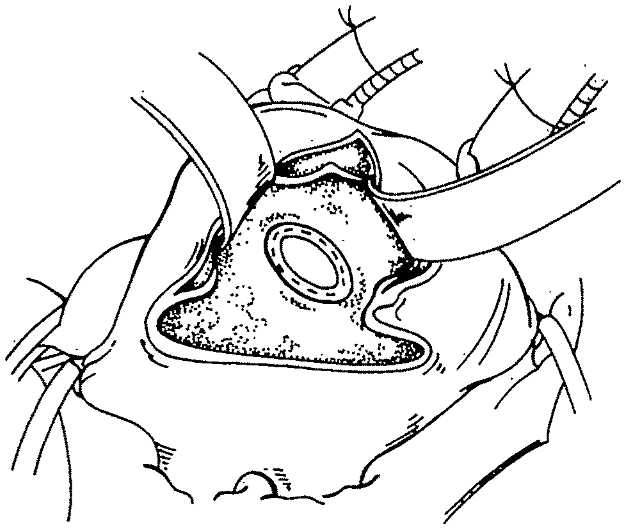

Fig 3. The retractors are moved inside the left atrium, achieving optimal exposure of the mitral area.

the interatrial groove and continuing into the anterior right atrial wall. A second continuous suture is applied to secure the left atriotomy (Fig 4).

Comment

This concept of an inverted T-shaped incision for mitral prosthetic replacement in the difficult small atrium has the advantage of being applicable after initial assessment of the adequacy (or inadequacy) of the standard approach. Application in all cases of difficult mitral prosthetic replacement is recommended.

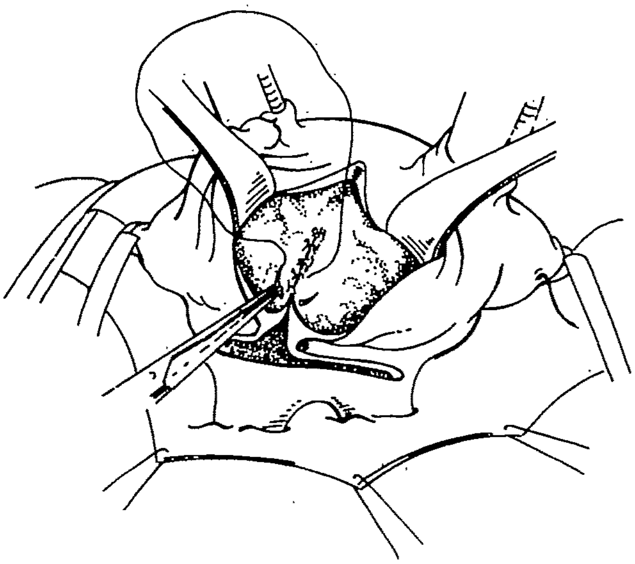

Fig 4. At the completion of the prosthetic replacement, the incision in the interatrial septum is secured with a suture including the interatrial groove and continuing into the right atrial wall. A second continuous suture is applied on the left atriotomy.

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Alternate Method for Revascularization of the Posterior Lateral Coronary Artery

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A technique is described for bypassing the posterior lateral branch of the right coronary artery by passing the vein graft beneath the inferior vena cava to avoid kinking of the graft.

(Ann Thorac Surg 1990;50:838)

The posterior lateral branch may originate from the right coronary artery at the crux and then course toward the posterior apical portion of the heart. This vessel supplies the diaphragmatic surface of the right and left ventricles and is usually parallel and posterior to the posterior descending coronary artery. A bypass graft to this vessel, when brought anterior to the inferior vena cava, is forced to bend acutely, which may result in kinking of the vein graft. The vein bypass graft may also kink if the length of the vein is overestimated. Underestimation of the length of the vein places excessive tension on the bypass conduit. To avoid these problems we place the vein bypass graft posterior to the inferior vena cava and anterior to the right atrium en route to the ascending aorta.

The distal coronary anastomosis is performed first during cardiopulmonary bypass and after cardioplegic arrest. The posterior lateral branch is exposed by an assistant elevating the heart and rotating it to the patient's left. A pack under the left atrium helps to maintain exposure. After construction of the distal anastomosis, the vein graft is filled with cold cardioplegic solution to make certain that there are no side branches or bleeding points in the portion of the vein that will be lying beneath the vena cava. The pericardium between the inferior vena cava and inferior pulmonary vein is divided. The vein graft is then directed inferior to the vena cava and anterior to the right atrium toward the ascending aorta (Fig 1). Just before performing the proximal anatomosis, the surgeon distends the vein with blood while determining the vein length. After application of a partial occluding clamp, a 4.5-mm opening is created on the anterolateral surface of the aorta, and the vein graft is sutured with 5-0 Prolene suture (Ethicon, Somerville, NJ) so as to direct the vein to the right. Suturing of the vein graft to the right atrium will prevent kinking if the graft is too long. This technique has been used in over 30 patients with no complications.

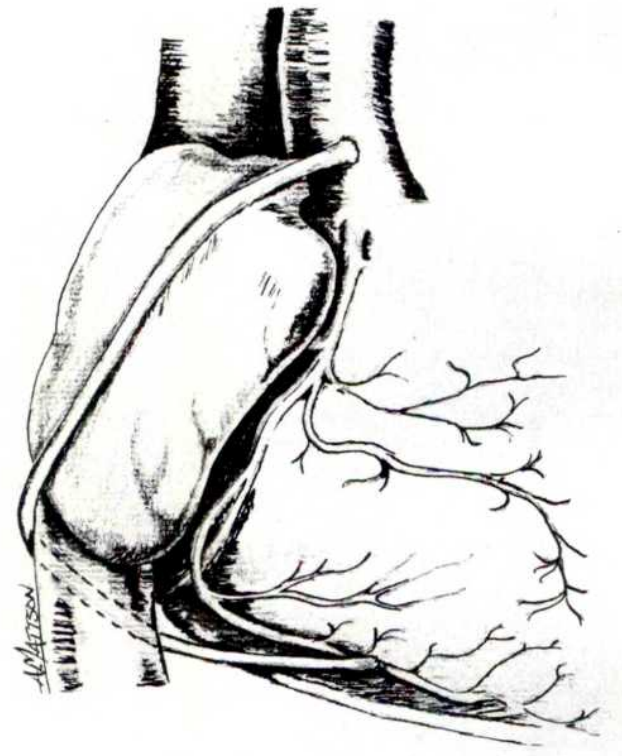

Fig 1. Course of the saphenous vein bypass graft anterior to the right atrium and inferior to (beneath) the inferior vena cava to a posterior lateral branch.

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Collis Gastroplasty: Origin and Evolution

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In 1957 J. Leigh Collis published his innovative operation for treating the difficult problem of the irreducible hiatal hernia, esophagitis, and stricture. The design of the operation was based on the relatively primitive understanding of hiatal hernia and the newly emerging concept of reflux esophagitis. A variety of antireflux operations by different surgeons emerged over the years to follow. The original Collis gastroplasty has been subsequently modified with the addition of both partial and complete fundoplication procedures. The place of the modified Collis gastroplasty-fundoplication operations in today's approach to the problems of hiatal hernia and gastroesophageal reflux disease remains unsettled.

(Ann Thorac Surg 1990;50:839–42)

An invitation to write a commentary on J. L. Collis' 1957 classic "An operation for hiatus hernia and short esophagus" [1] as a contribution to today's surgical management of hiatal hernia (HH) and gastroesophageal reflux disease awakens dormant memories of that remarkable period during the 1950s through which I lived as a thoracic surgeon. Today's younger thoracic surgeon need only read the discussions following Collis' 1957 presentation, which include operations such as moving the esophagus forward to the dome of the diaphragm for a shortened esophagus, interposing a segment of bowel containing the ileocecal valve to eliminate reflux, and pyloroplasty for promoting an empty stomach, to appreciate the advances since the 1950s.

After completion of my thoracic surgery residency in Ann Arbor almost four decades ago, I was fortunate to have been granted a fellowship at the Brompton Hospital for Chest Diseases, London, England. During this time I was privileged to visit with Mr Collis in Birmingham. This fellowship also afforded me the unique opportunity of spending time with two other pioneer English esophageal surgeons, P. R. Allison of Leeds and N. R. Barrett of London. These British surgeons played a major role in changing the established perception of the sliding HH from that of an anatomical mechanical condition to a functional physiologically based disorder with reflux esophagitis and its complications as the fundamental problem.

The 1957 Collis Operation

Collis sought and devised a relatively uncomplicated operation for the difficult HH with short esophagus, esophagitis, and stricture particularly in "the frail and aged." He wished to avoid the two extreme practices of either the risky, more complicated resectional operations of that day or the programs of endless esophageal dilations with ineffective medical management. Of the various factors thought to control gastroesophageal competence at that time, Collis believed "the acute angle of implantation of the esophagus into the stomach which in turn is produced by the normally functioning crural muscle . . . was the one which could be effective alone." Collis created a narrow, somewhat distally tapered tube along the proximal lesser curvature of the stomach that connected in continuity the shortened esophagus above with the stomach below (Fig 1). The gastric tube (gastroplasty), fashioned by suturing the stomach divided between two parallel clamps, met his two major objectives: (1) reestablishing the normal, more acutely angled (flap-valve) subdiaphragmatic junction with the stomach and (2) eliminating the irreducible stomach bulk from the hiatus. Collis also sutured the crura of the widened hiatus together anterior to the gastric tube displacing the tube further posteriorly, resulting in a more oblique angulation.

Collis used a thoracoabdominal incision with division of the costal arch. Today, the operation is generally carried out transthoracically. Subdiaphragmatic exposure can be obtained when needed by a peripheral circumferential opening of the diaphragm.

In his 1957 paper, Collis noted that the esophagus became lengthened after its transthoracic dissection and complete mobilization, now an established thoracic surgery principle: "In some cases it will be found that so much extra length of the esophagus can be obtained by this dissection that a standard repair can be done." This statement also carries an important implication for the modern esophageal surgeon, which is that one must be prepared to alter plans in response to unexpected operative findings.

Stricture and Dysphagia

Concerning peptic strictures, Collis wrote in 1957, "many strictures which are believed to be fibrous are, in fact, mainly produced by reflux spasm. In these cases when the irritation of reflux has been removed and the esophagitis has subsided the dysphagia will no longer be present." Today, there is general agreement that if a reflux stricture can be adequately dilated preoperatively

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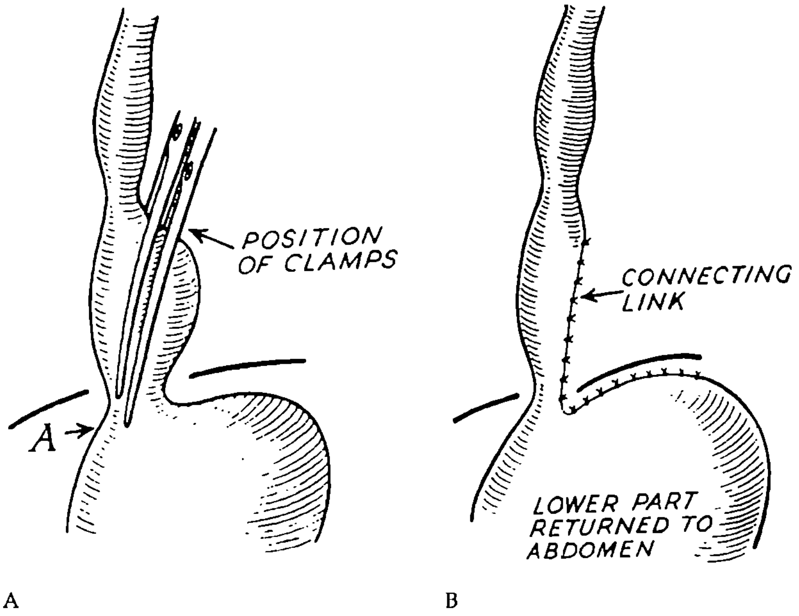

Fig 1. (A) Position of clamps on herniated stomach. (B) The newly created gastric tube (gastroplasty) connects the short esophagus and stomach beneath the diaphragm (see text). (Reproduced from [1] with permission from the publishers.)

or at operation, proper control of reflux should prevent stricture recurrence. Postoperative dilations may be required over a period of time for the occasional more severe stricture until the inflammatory reaction subsides. Resection of an esophageal stricture is now reserved for relatively uncommon situations such as an extremely resistant stricture associated with panmural fibrosis and inflammation, inadequate tissues for repair resulting from previously failed antireflux operations, and premalignant dysplastic changes associated with a Barrett esophagus.

Although Collis reported that 7 of his 8 postoperative patients had "excellent" results, all had dysphagia for weeks to several months after operation. It might be noted here that Collis did not have available the more effective flexible tapered-tip Maloney bougies nor modern balloon dilators. At that time it was also common HH surgical teaching to suggest that postoperative dysphagia was to be expected. Collis wrote, "some degree of immediate postoperative dysphagia is regarded as desirable and it is felt that if it is not present the operation has not been done properly." Today, surgeons try to avoid postoperative dysphagia by carefully tailoring the diameter of the hiatus and the antireflux procedure. A delicate balance must be achieved between maintaining normal food passage from above and preventing reflux from below.

Is Collis Gastroplasty a Barrett Esophagus?

Collis was aware that he was leaving "stomach" above the diaphragm proximal to the junction of tube and stomach. He defended this by citing references that this lesser curvature area of the stomach (Magenstrasse) used for his connecting tube was "devoid of peptic cells and relatively innocuous." Collis additionally drew on the analogy to those patients "often referred to as having gastric-lined esophagus and in whom symptoms are often slight." It is interesting to note that N. R. Barrett [2] also published his classic paper in 1957 that described a condition related to reflux esophagitis that came to bear his name. The Barrett esophagus was soon noted to add further complications [3], some of which may have a relationship to the Collis gastroplasty.

Skinner and Demeester [4, 5] have questioned whether the Collis gastroplasty might be an iatrogenic Barrett esophagus and carry the same biological potential for the development of malignancy as has been well documented in the acquired Barrett esophagus associated with chronic reflux esophagitis. A few patients found to have carcinoma in association with modified Collis gastroplasty for chronic esophagitis have been cited to support this contention. Pearson and associates [6], however, strongly question this assumption, suggesting that the tumor is related to the presence of an acquired Barrett mucosa rather than the gastric tube itself. This unsettled issue illustrates the need for thorough evaluation of all patients, particularly preoperatively, which would include at endoscopy circumferential brushings with washings for cytologic studies and multiple biopsies around and within strictures.

Collis Gastroplasty, Inert or Dynamic?

Both Skinner and Demeester consider the Collis gastroplasty to be an adynamic and inert tube. There is also a question as to whether the gastroplasty itself might act as an obstructing factor particularly in the presence of decreased esophageal motility. Cooper and co-workers [7] and Pearson and associates [6], however, have demonstrated that the gastroplasty has a synchronous function with the lower esophageal sphincter. Their manometric studies show that the gastroplasty responds to swallowing as well as to specific hormonal stimuli. The tone of the gastroplasty appears to be enhanced by the fundoplication. The exact function of the modified gastroplasty remains unsettled at this time.

Modified Collis Gastroplasty

The 1957 Collis gastroplasty by itself did not prove to entirely control reflux esophagitis. About that time, experimental studies were published that showed that creating an oblique gastroesophageal angle (flap-valve), even if exaggerated, failed to prevent reflux unless the opposite part of the esophagus was effectively buttressed, supported, or wrapped [8]. It should be recalled that the question of the existence of a functioning lower esophageal sphincter, not demonstrable anatomically, was not finally established until the development of new manometric techniques at the end of the 1950s [9]. The decade of the 1960s was a time when various buttressing and wrapping antireflux operations became established.

In 1961 Nissen [10] published an important clinical experience treating HH with a complete 360-degree gastric wrap around the esophagus termed a fundoplication. Belsey, after trials and careful study, published his Mark IV operation [11], a transthoracic telescoping procedure with stomach partially wrapped around the esophagus all anchored beneath the diaphragm.

Collis Gastroplasty with Belsey Operation

Pearson and associates of Toronto [12] deserve credit for combining the Collis gastroplasty with the Belsey procedure. This made the gastroplasty a more effective antireflux operation and addressed the long-term unsatisfactory results with the Belsey repair alone in the presence of subtle esophageal shortening, thereby eliminating the factor of tension. Pearson's combined Collis-Belsey operation also helped popularize the transthoracic approach to HH and reflux esophagitis.

The Collis-fundoplication repairs are used for a select group of patients with a shortened esophagus, stricture, transmural ulcerative esophagitis, large combined sliding and paraesophageal HH difficult to reduce without tension, and a previously failed antireflux operation. The Toronto Collis-Belsey series has been followed up for more than 15 years with excellent subjective and objective results in approximately 90% of their patients. The outcome is less favorable after multiple previous antireflux operations, advanced scleroderma, and certain esophageal motility disorders. It is probably contraindicated in the presence of achalasia, difficult strictures, and multiple destructive previous HH-antireflux operations.

Collis-Nissen Fundoplication

Orringer and Sloan of Ann Arbor [13] discontinued using the Collis-Belsey procedure because of an unacceptably high failure rate and changed to the Nissen complete 360-degree antireflux gastric wrap. Not only did the gastroplasty create a longer pseudoesophagus, but tension-free sutures could be placed in the gastric tube rather than the often tenuous and friable distal esophagus. The Ann Arbor group [14] has reported excellent results with the Collis-Nissen procedure comparable with the Toronto Collis-Belsey series for roughly the same type of patient with similar indications and contraindications. Both the Toronto and Ann Arbor series represent the largest, longest followed and most carefully studied consecutive series of patients having combined Collis gastroplasty and fundoplication operations. One can only speculate on what subtle differences in case selection, surgical technique, and follow-up methodology play in the results from these two highly competent centers of esophageal surgery, one using the Collis-Belsey and the other the Collis-Nissen gastroplasty. The Nissen fundoplication may offer better reflux control but it carries the potential for producing dysphagia and inability to belch or vomit. This has prompted the call for a shorter and looser gastric wrap. The Belsey-type repair carried out with gastroplasty, when adequate stomach is available, would be preferable in the presence of an esophageal motility disorder.

Intrathoracic Fundoplication Procedures

Another approach to the short esophagus and irreducible HH is an intrathoracic Nissen fundoplication left above the diaphragm. When the HH was associated with a tight stricture, Thal and colleagues [15] incised the stricture longitudinally suturing the side of the stomach as a patch over the esophageal opening. An intrathoracic Nissen-type fundoplication was subsequently added to control reflux. This so-called Thal-Nissen operation [16] has gained very limited acceptance.

Fundoplication procedures leaving the stomach in the chest expose the patient to the well-documented serious complications associated with a paraesophageal HH. The Collis lengthening-antireflux concept is a preferable approach. With a long, upper thoracic or intractable esophageal stricture, resection would be indicated in the good-risk patient.

The Stapling Device and Modified Collis Gastroplasty

Introduction of the gastrointestinal anastomosis stapling device has simplified creation of the Collis gastroplasty. Unfortunately, the gastroplasty has probably been unnecessarily used in uncomplicated cases where attention to established principles of adequate esophageal mobilization and complete dissection would permit a tension-free antireflux procedure without the need to add the gastroplasty. The stapler has also been used for forming a gastric tube without cutting and oversewing the stapled edges, the so-called uncut gastroplasty [17]. The undivided stapled stomach does not eliminate staple cut-through and the occasional leak and fistula associated with the Collis gastroplasty.

Conclusions

The Collis gastroplasty combined with an antireflux procedure is an effective operation for a select set of adult patients. With the increasing availability of new reflux measuring techniques in competent gastroesophageal testing facilities throughout the country, the place of the modified Collis gastroplasty operation should become delineated more precisely than the current situation, in which the operation is practically never used by some or almost routinely used by others. Furthermore, a new generation of endoscopically trained gastroenterologists armed with an ever-increasing arsenal of more sophisticated drugs for managing gastroesophageal reflux disease will challenge the practicing surgeon to produce standardized statistically valid data to support the place of modern Collis gastroplasty. The esophageal surgeon of tomorrow will certainly be under stricter scrutiny in selecting the appropriate operation for each patient in a changing population with more complex reflux disease.

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Blood Conservation in Cardiac Surgery

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We reviewed current blood conservation techniques and their use in cardiac surgery. Avoidance of aspirin preoperatively is an important blood conservation measure. Patients scheduled for an elective operation should participate in autologous predonation programs. With careful monitoring, patients with major coronary artery disease can safely donate blood preoperatively. Intraoperative processing of blood withdrawn before cardiopulmonary bypass provides autologous platelet-rich plasma for infusion after reversal of heparin sodium. Blood collected from the field during operation and blood remaining in the oxygenator after bypass can also be processed to yield washed and concentrated red blood cells for reinfusion. Randomized, prospective studies document that postoperative autotransfusion is both safe and effective in reducing homologous blood use. Aprotinin reduces plasma protein activation and platelet damage during bypass. The integration of available blood conservation techniques into a comprehensive program combined with careful consideration of the indications for transfusion may allow more patients to avoid transfusion entirely.

(Ann Thorac Surg 1990;50:843–51)

Homologous blood transfusions during and after cardiac operations subject the patient to the risks of transfusion reactions, sensitization to blood products, and transmission of infectious agents. The incidence of posttransfusion hepatitis in the United States is approximately 1%, as estimated by the National Institutes of Health Consensus Conference on Perioperative Red Blood Cell Transfusion [1]. The recent development of a test for the virus that causes non-A, non-B hepatitis (hepatitis C) should further reduce this incidence. Although hepatitis C is the most common blood-transmitted disease in industrialized nations, public attention has focused on the relationship between blood transfusions and acquired immunodeficiency syndrome. In the United States, the risk of contracting acquired immunodeficiency syndrome from a blood transfusion has been estimated at 1:40,000 to 1:1,000,000 [1–3]. Nevertheless, public concern over the safety of homologous blood transfusions and the national mandate to provide more cost-effective medical care have resulted in renewed interest among cardiac surgeons in blood conservation. The purpose of this article is to review current blood conservation techniques as they apply to cardiac surgery. These techniques can be combined into a comprehensive blood conservation program.

Preoperative Blood Conservation Techniques

The general topic of autologous transfusion has been exhaustively reviewed by AuBuchon [4]. Preoperative donation of blood by patients scheduled for elective surgical procedures provides blood that ordinarily would not be available and avoids many of the hazards associated with transfusion therapy. A recent national multicenter study [5] of autologous blood donation before elective cardiac and noncardiac operations determined that only 5% of the eligible patients actually predeposited blood. If all eligible patients had predeposited autologous blood, it was estimated that they could have supplied blood for as much as 72% of their own subsequent transfusion requirements.

The reasons cited in the study for the underutilization of predonation programs included the added inconvenience of predonation to patients, physicians, and blood centers. In addition to logistical problems relating to donation and storage of autologous blood, concerns about patient safety continue to limit the number of patients scheduled for cardiac operation who participate in predonation programs. Often these patients are considered ineligible for participation because of the fear of precipitating myocardial ischemia or infarction at the time of blood donation.

The results of numerous studies [6–9] tend to refute these concerns. Autologous blood collection has been performed safely in patients with severe heart disease (including coronary artery disease), in children, and in the elderly. Mann and co-workers [6] presented a series of 342 high-risk patients who participated in an autologous blood donation program. Patients with frequent angina were accepted as autologous donors, but all those with preinfarction angina were excluded. Units of blood were collected with a minimum interval of 4 days between donations. All patients received oral iron supplementation. Vital signs and serum hemoglobin level were determined before each donation. Electrocardiographic monitoring was not performed. Scheduled donations were deferred temporarily because of anemia in 3% to 7% of patients. The incidence of light-headedness and fainting was 2% to 5%, equivalent to that seen in normal volunteer

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blood donors. Despite this, no clinical ischemic episodes were observed.

Three more recent reports [7–9] also conclude that predonation is safe for patients with cardiac disease and effective in reducing homologous blood use, although they were not randomized, prospective studies. Love and associates [7] compared patients requiring cardiac operations who predonated blood with matched controls. Patients with aortic stenosis and critical coronary lesions were excluded. All patients received ferrous sulfate. They donated an average of 1.97 units of blood over 18 days. The mean decrease in hemoglobin level was 22 g/L (2.2 g/dL). No complications of predonation were reported, despite a history of myocardial infarction in 62% of the patients. The mean number of homologous units transfused in the predonation group was 1.1 compared with 2.05 units in the control group ($p < 0.006$). A total of 63.8% of the predonation group avoided homologous transfusion compared with only 37.9% of the control group ($p < 0.02$).

Britton and associates [8] also reported the results of a predonation program in patients undergoing elective cardiac procedures compared with a group of concurrent controls. Blood was collected in the hospital blood bank under physician supervision. Homologous blood products were avoided in 75% of patients in the predonation group compared with 21% in the control group. An average of 4.1 units of blood was predonated (at a rate of 1 unit/week), but 12 patients (11.5%) had to discontinue donations. Donations were discontinued because of increasing angina in 10 patients (2 required hospitalization) and because of anemia (hematocrit < 0.33) in the other 2 patients. Four patients underwent early operation for an increase in symptoms during the donation period. However, during the same period, 4 patients who had not participated in predonation also required an earlier surgical date because of increasing symptoms. Britton and associates concluded that, although patients with severe coronary artery disease are at risk for experiencing increasing symptoms during the time required for donation, this risk did not appear to be related to the predonation process per se.

Owings and colleagues [9] reviewed the records of 107 patients who predonated an average of 3.0 ± 1.5 units of blood before undergoing cardiac operation. All patients received oral iron. Normal saline solution (500 mL) was infused during each donation to maintain intravascular volume. Dizziness occurred in 1 patient, and angina requiring admission occurred in another (2/326 total donations). Importantly, 73% of patients in the predonation group avoided homologous blood transfusion compared with only 18% of concurrent controls.

The American Association of Blood Banks has established guidelines for predonation programs [10]. There is no upper age limit for donors. No more than 450 mL (or 12% of estimated blood volume, whichever is less) should be withdrawn at a single donation. Some authorities [9] recommend isovolemic replacement with saline solution during each donation. The hemoglobin concentration should be 110 g/L (11 g/dL) or greater (hematocrit, 0.34 or greater) at the time of donation. Donations should be no more frequent than every 3 days, the time required for plasma volume to return to normal. Because body iron stores can limit the rate of erythropoiesis, the American Association of Blood Banks recommends that patients undergoing frequent phlebotomy receive oral iron supplementation (ferrous sulfate, 325 mg, three times daily). Oral iron is as effective as parenteral iron in this application. Iron supplementation should be started at least 1 week before the first donation and continued for several months after the last donation.

Intraoperative Techniques of Blood Conservation

Intraoperative blood conservation techniques are routinely used in cardiac surgery [11]. These techniques include (1) intraoperative phlebotomy before cardiopulmonary bypass (CPB); (2) use of cystalloid oxygenator prime to achieve normovolemic hemodilution; (3) intraoperative retransfusion of all blood removed by cardiotomy suction; (4) reinfusion of all blood remaining in venous tubing and the oxygenator after CPB; (5) acceptance of postoperative anemia in patients in hemodynamically stable condition; and (6) adequate hemostasis. These techniques must be carefully applied in the individual clinical situation to maximize the benefits and minimize the risks of blood conservation.

Withdrawal of Autologous Blood Before Cardiopulmonary Bypass

The damaging effect on blood of CPB results in defective hemostasis. Excessive bleeding can be treated with fresh frozen plasma, platelet transfusions, and fresh blood. Dodrill and associates [12], in 1957, first reported the use of blood collected intraoperatively before CPB to treat excessive bleeding after CPB, a technique subsequently studied and adopted by other groups [13, 14]. Blood is collected before heparinization and CPB and is stored at room temperature in standard anticoagulant (acid-citrate-dextrose or citrate-phosphate-dextrose) [13–16]. Usually, 15% to 20% of total blood volume is collected (approximately 2 units). Isovolemic replacement with crystalloid solution is performed, resulting in hemodilution. Therefore, patients with substantial anemia preoperatively are not candidates for this procedure. We and others [16] would also exclude patients with aortic stenosis, left main disease, or unstable angina. Autologous blood is returned to the patient after cessation of CPB, neutralization of heparin, and control of surgical bleeding sites. In addition to providing autologous platelets and clotting factors after CPB, the technique reduces the red cell mass of blood shed during the operation.

Many groups [12–16] have documented 20% to 58% decreases in homologous blood requirements when autologous blood is transfused intraoperatively after CPB. The decreased bleeding has been attributed to clotting factors and platelets supplied by the autologous blood, which has been spared exposure to the extracorporeal circuit. Lavee and colleagues [17] have claimed that the hemostatic effect of 1 unit of fresh whole blood is at least equal to that of 10 units of platelets.

Cohn and co-workers [18] demonstrated that platelet-rich plasma (PRP) could be separated from autologous blood collected intraoperatively before CPB. The advantage of this technique is that the blood components most susceptible to the damaging effects of CPB (platelets and clotting factors) are preserved, and red blood cells are separated and immediately reinfused to provide oxygen-carrying capacity during CPB. In this study, patients receiving autologous PRP had higher postoperative platelet counts. However, there was no difference in the amount of homologous blood transfused to these patients compared with those not receiving autologous PRP. This may be due to the fact that relatively small amounts of PRP (mean amount, 337 mL/patient) were reinfused.

Giordano and associates [19] reported their experience with the intraoperative withdrawal and processing of whole blood into a PRP fraction. An average yield of 1.0 to 2.0 × 10^{11} platelets in 500 mL of plasma was obtained. Their results demonstrated a decrease in homologous blood use and bleeding compared with historical controls when intraoperative PRP collection was combined with standard blood conservation techniques.

More recently, Jones and associates [20] reported the results of a randomized prospective clinical trial of the effects of intraoperative plasmapheresis on 100 consecutive patients undergoing elective coronary artery operations. Approximately 1,000 mL of autologous PRP containing an average of 2.5 ± 0.35 × 10^{11} platelets were reinfused per patient. Significant reductions in red cell mass lost perioperatively and mean number of units of homologous blood products transfused were observed. Most importantly, the percentage of patients not receiving homologous blood was greater in the PRP group compared with controls (66% versus 32%; $p = 0.001$). Strict indications for transfusion were established and supposedly followed for both groups to ensure that the control group was not simply overtransfused.

Further investigation is needed to determine the role of intraoperative platelet plasmapheresis in a blood conservation program. For example, it is not clear that routine processing of blood collected before CPB into PRP, with immediate reinfusion of red cells, is advantageous except in those patients who would otherwise not be candidates for intraoperative blood donation because of low preoperative hemoglobin levels.

Use of Crystalloid Oxygenator Prime

The use of substantial levels of hemodilution with CPB has been standard procedure since the early work of several groups [21–23] demonstrated that crystalloid solutions could serve as a complete prime for oxygenators. Generally, normovolemic hemodilution results in a hematocrit of 0.20 to 0.30, a level determined to combine decreased viscosity with acceptable oxygen-carrying capacity. This resulted in a major (30% to 50%) decrease in transfusion requirements for procedures requiring CPB [22–24]. Besides conserving blood, hemodilution is associated with improved postoperative pulmonary and renal function as a result of decreased sludging and aggregation of formed blood elements [24–26]. Intraoperative and postoperative metabolic acidosis was less severe when hemodilution was employed, suggesting improved perfusion during CPB [26]. The use of more extreme degrees of hemodilution has been reported [27] but is not routine.

The main disadvantage of hemodilution is an increase in extracellular fluid, mostly in the interstitial compartment [28]. This fluid leaves the intravascular space during CPB and reenters during the first 4 postoperative days, making fluid restriction and diuretic administration necessary [28]. However, the overall benefit of a moderate degree of hemodilution outweighs the problem of increased interstitial fluid.

Intraoperative Blood Salvage

After heparinization, blood shed at operation is routinely returned to the pump-oxygenator and reinfused. Particulate matter and bubbles must be removed. For these reasons, blood collected from the field passes through a defoamer and filtered reservoir before reinfusion. However, it is well documented that aspiration of blood from the pericardium and intracardiac chambers damages blood elements. Use of cardiotomy suction is discontinued when protamine sulfate is administered; however, a substantial amount of blood loss can still occur after this time.

To maximize intraoperative blood salvage, many groups have replaced high-power discard or wall suction with a blood-processing system. The Haemonetics Cell-Saver System (Haemonetics Corporation, Braintree, MA) is typical of the devices in current use. It removes blood from the field using the wall vacuum as a source of suction. An anticoagulant (heparin or citrate) is delivered through double-lumen tubing to the suction catheter and mixes with aspirated blood en route to a standard cardiotomy reservoir containing a 120-μm filter. Therefore, the system can be used before heparin is administered and after heparin is reversed with protamine. The collected blood-anticoagulant mixture can then be delivered to a centrifuge bowl where red blood cells are separated from plasma and other formed elements. The red blood cells are washed with saline solution, resulting in a product with a hematocrit of approximately 0.60 and ready for immediate reinfusion. The product is largely free from platelets and clotting factors, but will have had 85% of the heparin removed by the centrifugation and washing cycles [4]. Rapid reinfusion of blood resulting from a major bleeding site is not practical with these devices; therefore availability of standard cardiotomy suction is still advisable.

Because the amount of blood lost before and after CPB in most patients can be expected to be minimal, the benefit of using a Cell Saver to replace standard wall suction during primary coronary artery operations has been questioned. In a group of 20 patients undergoing routine cardiac surgical procedures, Winton and co-workers [29] found the mean volume of packed red cells salvaged intraoperatively with the Cell-Saver System to be only 105 ± 88 mL. These authors did not use the Cell Saver to process the residual oxygenator contents after CPB, however.

After CPB, a substantial number of the patient's red blood cells are distributed in the extracorporeal circuit, ie, the oxygenator and blood tubing. Maximum blood conservation requires routine drainage of all venous blood into the oxygenator with reinfusion of oxygenator contents after decannulation. Because this blood is dilute, excessive fluid can be administered along with any heparin present in the extracorporeal circuit. Diuretics can be given intraoperatively to promote a brisk diuresis. Alternatively, oxygenator contents can be hemoconcentrated using a cell separator and then reinfused. By combining intraoperative salvage of blood shed from the operative field with processing of the residual oxygenator contents after CPB, several groups [30–35] were able to demonstrate not only significant reductions in blood usage but also cost-effectiveness [33, 35] of currently available systems.

Boldt and associates [36] compared hemofiltration with centrifugation as an alternative method of concentrating the blood remaining in the extracorporeal circuit before it is reinfused. Hemofiltration was performed after CPB by pumping the remaining blood in the venous reservoir through the hemofilter with a roller pump. The amount of blood produced by hemofiltration after CPB was greater than that produced by cell separation, and hemofiltration was less time-consuming. The mean hemoglobin level in blood concentrated by hemofiltration, however, was lower than that in blood prepared by cell separation (mean level, 161 ± 19 g/L [16.1 ± 1.9 g/dL] versus 193 ± 18 g/L [19.3 ± 1.8 g/dL]; $p < 0.05$). The mean platelet count of the processed blood was greater after hemofiltration ($381 \pm 85 \times 10^6$/L) than after cell separation ($74 \pm 28 \times 10^6$/L) ($p < 0.05$). The free hemoglobin and potassium levels were elevated in hemofiltered blood, but elevated free hemoglobin and potassium levels were not observed in patients who had autotransfusion with this blood. Postoperative blood loss (through the first postoperative day) was similar in patients who received blood processed by cell separation or hemofiltration (mean loss, 547 ± 113 mL versus 532 ± 265 mL). Both techniques appear to be acceptable methods for concentrating residual oxygenator contents before reinfusion. Centrifuge-based systems can be used throughout the operation to separate blood collected intraoperatively and therefore may be more generally useful than hemofiltration systems.

Postoperative Techniques of Blood Conservation

Postoperative Autotransfusion of Shed Mediastinal Blood

Reports of reinfusion of blood lost from an injury or surgical procedure have appeared in the medical literature since the early 1800s [37]. Autotransfusion with blood shed from a traumatic hemothorax was first reported by Elmendorf [38]. However, it was Schaff and associates [39] who first reported autotransfusion of blood collected from the mediastinum after elective cardiac procedures. Mean blood loss after a cardiac operation ranges from 765 to 1,250 mL per patient [11, 39, 40]. This represents an important source of blood replacement if formed elements and coagulation factors are present at nearly normal levels.

Several groups [11, 39–42] have studied the composition of blood collected from the mediastinum after cardiac operations. The mean hematocrit ranges from 0.20 to 0.25. Most groups have found elevated levels of plasma free hemoglobin in drainage blood, presumably as a result of red blood cell hemolysis. However, Symbas [43] showed that the half-life of autotransfused red blood cells collected from the pleural space in an experimental model of hemothorax was similar to that of venous blood from a control group, a finding suggesting that red cell injury from collection and retransfusion is minimal. Platelets are present in reduced numbers in blood collected from the mediastinum and are probably not functional. Raines and associates [44] observed a decrease in the function of platelets collected from the operative field during abdominal vascular procedures.

Solem and co-workers [40] found that levels of electrolytes and albumin in mediastinal drainage were similar to those in normal serum. Depletion of fibrinogen is characteristic of blood collected from the mediastinal and pleural spaces. Most groups measured major reductions in the activity of factors II, V, VII, IX, and X in blood collected from similar autotransfusion systems [41, 42, 45] along with an increase in fibrinolytic breakdown products [41, 42, 46]. An active process of clot formation with consumption of coagulation proteins occurs in the mediastinum and pleural spaces. This process is responsible for the fact that shed blood usually does not clot.

Postoperative Autotransfusion Systems

Clinical studies of autotransfusion after cardiac operation have been conducted using commercially available systems or modifications of these systems. The most commonly used system was originally developed by Noon [47] in conjunction with Sorenson Research Corporation (Salt Lake City, UT). This system consists of two collection reservoirs connected in series and housed within a rigid cannister, which is then attached to a -20-cm H_2O vacuum source. As blood is collected in the upper reservoir, it passes through two 170-μm screens before entering the lower reservoir. The lower reservoir is disconnected, and blood is reinfused by gravity through a 40-μm Pall filter.

Systems incorporating commercially available cardiotomy reservoirs have been used clinically [48]. At the conclusion of the operation, the cardiotomy reservoir is removed from the bypass circuit, chest tubes are attached to the inlet ports of the cardiotomy reservoir, negative pressure is applied, and drainage blood is collected. All collected blood is continuously reinfused as long as the patients has substantial chest tube drainage. The risk of contamination may theoretically be decreased by maintenance of a closed system [48].

Clinical Experience With Autotransfusion

The efficacy of postoperative autotransfusion systems in reducing the amount of homologous blood transfused after cardiac operations has been reported by many authors. Three of these studies [11, 39, 42] were conducted in a randomized, prospective fashion and are reviewed here.

Schaff and associates [39] randomized 114 adults undergoing a cardiac procedure, including valve replacement, to an autotransfusion group (n = 63) and a control (no autotransfusion) group (n = 51). The Sorenson autotransfusion system was used. If the rate of mediastinal bleeding was slow and 4 hours passed without a 400-mL volume being collected in the autotransfusion system bags, the blood was not reinfused because of the possibility of bacterial contamination. Of the 63 patients randomized to the autotransfusion group, 33 (52.3%) actually received a transfusion of shed mediastinal blood. There was no difference between the two groups with regard to preoperative characteristics, types of operations performed, or total postoperative bleeding (autotransfusion group, 813 ± 121 mL/m^2, and control group, 711 ± 93 mL/m^2; p = not significant). Mean postoperative blood replacement was similar in the two groups (autotransfusion group, 4.3 ± 0.6 units/patient, and control group, 4.8 ± 0.6 units/patient; p = not significant). However, requirements for transfusion of homologous blood were reduced by 50% in the autotransfusion group (autotransfusion group, 2.4 ± 0.3 units/patient, and control group, 4.8 ± 0.6 units/patient; $p < 0.005$). The average amount of mediastinal blood autotransfused in the experimental group was 1.9 ± 0.4 units/patient.

Thurer and colleagues [11] studied 113 consecutive adult patients undergoing a cardiac operation, excluding valve replacement. There were 54 patients randomized to the postoperative autotransfusion group and 59 patients randomized to the control group. The Sorenson autotransfusion system was used in this study. Shed blood that was not used during any 4-hour period was discarded. Forty-five patients in the autotransfusion group were actually given transfusions of shed blood (mean amount, 523 mL/patient). The mean amount of homologous blood transfused postoperatively was similar for each group (autotransfusion group, 510 mL/patient, and control group, 534 mL/patient). However, the total amount of blood transfused postoperatively (homologous and autotransfusion) was greater in the autotransfusion group. The reason for the increased transfusion requirement in the autotransfusion group is not clear, as the initial postoperative hematocrit, total mediastinal drainage, and coagulation profiles measured in both groups were similar.

Adan and associates [42] performed a randomized prospective study of the efficacy of autotransfusion in two groups of 25 adult patients undergoing routine coronary artery bypass grafting. The Sorenson autotransfusion system was used. Adan and colleagues noted that, although a 50% reduction in homologous blood use was achieved, they were not able to avoid donor blood use completely. Blood was transfused only if a minimum of 250 mL was collected in a 4-hour period. Bleeding rates were slower in 10 of the 25 patients in the autotransfusion group who, therefore, received no mediastinal drainage blood. Furthermore, clot formation blocked the filters contained in the system in 2 of 25 patients who bled profusely, preventing use of the autotransfusion system in patients who needed it the most. Adan and co-authors therefore questioned the value of the routine use of postoperative autotransfusion.

Although the data from the study by Thurer and co-workers [11] are difficult to interpret, the studies reported by Schaff and associates [39] and by Adan and colleagues [42] document decreased use of homologous blood with the use of a commercially available postoperative autotransfusion system. Neither group was able to completely avoid the use of homologous blood. Schaff and associates used a postoperative hematocrit of less than 0.35 as an indication for transfusion, and Adan and co-authors accepted postoperative hematocrits of 0.25 to 0.30 in patients in hemodynamically stable condition. Adan and associates also observed practical problems with autotransfusion. Despite these problems, two of the three randomized studies documented decreased use of homologous blood when routine postoperative autotransfusion was adopted.

Safety of Postoperative Autotransfusion

Potential complications of postoperative autotransfusion can be categorized into hematologic (coagulation abnormalities), infectious, and renal. No differences in platelet count, free plasma hemoglobin, prothrombin time, partial thromboplastin time, euglobulin lysis time, fibrinogen, factor VII, or factor V were noted at any period postoperatively when patients receiving mediastinal drainage blood were compared with those who did not [11, 39, 41, 49]. Sensitive markers of systemic fibrinolysis such as fibrinopeptide A or B-beta 14–42 peptide were not elevated in patients receiving postoperative autotransfusions [41]. The mean volume of shed mediastinal blood that was retransfused in these studies varied from 500 mL to almost 2 L. No difference in the incidence of postoperative infection was observed when patients receiving reinfusions of mediastinal drainage were compared with controls [11, 39, 49]. In no patient with culture-positive mediastinal drainage did either clinical infection or positive blood cultures develop [11, 39]. In addition, no important differences in blood urea nitrogen or creatinine levels were observed in patients receiving postoperative autotransfusion compared with those who did not [11, 41].

Pharmacological Agents

Abnormal bleeding after CPB is the subject of a number of excellent reviews [50, 51]. Control of bleeding that occurs after CPB begins with effective heparin neutralization. The details of inducing, maintaining, and neutralizing heparin anticoagulation have also recently been reviewed [52]. The focus of the remainder of this review will be the pharmacological treatment of bleeding abnormalities occurring after CPB despite effective heparin neutralization.

Numerous studies have identified specific hemostatic defects produced by the contact of blood with the surface of the extracorporeal circuit. The most important is a qualitative defect in platelet function [53]. Thrombocytopenia plays a lesser role in bleeding abnormalities developing after CPB [50, 54]. The precise nature of the platelet defects that occur during CPB are not completely understood. Platelet dysfunction can occur as a result of either platelet activation with granule release and depletion of important functional components [55, 56] or acquired platelet membrane abnormalities [57, 58]. In addition to platelet dysfunction, activation of the plasma proteins that constitute the fibrinolytic, coagulation, and complement systems occurs during CPB [50, 57]. Postoperative elevation of plasma levels of C3a, a breakdown product of complement and an indicator of plasma-protein activation, has been identified as a risk factor for abnormal bleeding [59]. Although evidence for increased fibrinolytic activity during CPB exists, most authors [50, 60] believe this plays a minor role in bleeding after CPB. Therefore, current strategies directed at correcting the hemostatic defect after CPB use pharmacological agents to prevent platelet and generalized plasma-protein activation or to improve platelet function.

Avoidance of preoperative administration of aspirin is one way to preserve perioperative platelet function. Aspirin is commonly administered preoperatively to patients undergoing coronary artery bypass grafting to improve vein graft patency rates. However, a multicenter, randomized, prospective study [61] of antiplatelet agents administered to patients undergoing coronary bypass procedures found that perioperative blood loss and the number of reoperations for bleeding were significantly less in patients in whom aspirin was first started 6 hours postoperatively compared with patients in whom aspirin was administered preoperatively. This same study implied that vein graft patency rates are equivalent whether aspirin is started preoperatively or as late as 6 hours postoperatively. These data suggest that avoidance of preoperative aspirin administration will decrease the incidence of postoperative bleeding necessitating reoperation without compromising vein graft patency. It should be emphasized that aspirin use must be stopped several days preoperatively because of its irreversible effects on platelet function. However, because of the many indications for aspirin administration in patients with heart disease (in the treatment of acute ischemia or myocardial infarction [62] as an adjunct to thrombolytic therapy [63]), avoidance of aspirin preoperatively may be impractical for many patients.

Teoh and colleagues [64] performed a prospective randomized study of 58 patients undergoing elective coronary bypass procedures to evaluate postoperative blood loss and transfusion requirements in patients given dipyridamole versus control. One group was given oral dipyridamole (100 mg four times a day) starting 2 days before operation, a second group was given intravenous dipyridamole ($0.24 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$) beginning 22 hours before operation and continuing until the first postoperative morning, and a third group received no dipyridamole. Postoperative blood loss was significantly reduced (42% to 46%) in both groups receiving dipyridamole compared with controls, but was not significantly different between patients administered intravenous or oral dipyridamole. Platelet counts postoperatively were greatest in the intravenous group, intermediate in the oral group, and lowest in the control group. In view of the favorable influence on blood conservation demonstrated by Teoh and associates, further study of preoperative oral dipyridamole is warranted in patients undergoing coronary artery operations.

Because plasma proteins and enzyme systems are activated or adsorbed by contact with artificial surfaces, other groups have studied the administration of protease inhibitors as a means of reducing bleeding after CPB. In a randomized study, van Oeveren and associates [65] studied the effect of aprotinin administration before and during CPB in 11 patients. Compared with controls, the aprotinin-treated patients were able to maintain platelet numbers during CPB, although the numbers decreased, as did platelet counts in control patients, when protamine was administered. The measured blood loss and the transfusion requirement were significantly lower in the aprotinin-treated patients compared with the control group. Levels of complement breakdown products were similar in both groups. Aprotinin appeared to inhibit fibrinolysis during CPB, however. Van Oeveren and associates concluded that aprotinin preserved platelet function by preventing the exposure of platelet receptors, an initial step in platelet activation, by proteases such as plasmin.

Bidstrup and co-workers [66] reported the results of a placebo-controlled, randomized, double-blind study of 80 patients undergoing primary coronary artery bypass grafting who received either aprotinin or saline solution during the procedure. Platelet counts measured perioperatively did not differ between the two groups, but postoperative template bleeding time rose 6.2 ± 2.1 minutes in the placebo group compared with 1.5 ± 1.1 minutes in the aprotinin group. In addition, the total mediastinal drainage was significantly reduced in the aprotinin group compared with the placebo group (mean drainage, 309 ± 133 mL versus 573 ± 166 mL; $p < 0.01$). Aprotinin also significantly reduced blood loss in 11 patients undergoing reoperation after a previous median sternotomy. The findings reported by van Oeveren and associates [65] and Bidstrup and colleagues [66] demonstrate that aprotinin reduces blood loss after CPB. However, aprotinin is not yet available in the United States.

Desmopressin acetate (DDAVP) has shortened bleeding time and reduced surgical blood loss in patients with conditions associated with platelet dysfunction, such as uremia and aspirin ingestion [67]. Given these facts, several groups have studied the ability of desmopressin acetate to reverse platelet dysfunction after CPB.

Salzman and associates [67] reported the results of a randomized prospective study of the use of desmopressin acetate in patients undergoing operation for valvar heart disease, atrial septal defect, and repeat coronary artery procedures. The drug was not used in patients undergoing uncomplicated primary coronary artery procedures. Desmopressin acetate was administered intravenously at a dose of 0.3 μg/kg of body weight over 15 minutes at the conclusion of CPB. Total blood loss (at operation and in the first 24 hours) was 2,210 ± 141 mL in the group given a placebo and 1,317 ± 486 mL in the group given desmopressin acetate ($p = 0.001$). The volume of blood transfusion in the first 3 days was smaller in the group given desmopressin acetate (2.6 ± 2.1 units versus 3.7 ± 3.3 units; $p = 0.079$). Patients who received desmopressin acetate had a significantly higher platelet count 90 minutes later than did those who received the placebo (116,630 ± 43,600 versus 95,760 ± 35,920; $p = 0.04$). Plasma levels of von Willebrand factor were higher after desmopressin acetate than after a placebo. The patients with the most bleeding had relatively low levels of von Willebrand factor before operation, a finding suggesting a role for this factor in the hemorrhagic tendency induced by CPB. No complications of desmopressin acetate administration were identified.

The use of desmopressin acetate was not found to be of value in a prospective, randomized, double-blind trial involving 60 children undergoing cardiac operations [68]. It was also found to be of no value in reducing blood use in a double-blind, randomized, placebo-controlled trial of 150 adult patients undergoing primary coronary artery bypass grafting [69]. Despite the study by Salzman and colleagues [67], we do not recommend routine use of desmopressin acetate until the results of additional clinical trials become available.

Conclusion

One of the primary goals of the surgeon should be to avoid transfusion entirely. This can best be achieved from within the framework of a comprehensive blood conservation program. As part of such a program, however, the cardiac surgeon must apply his or her knowledge of the alternatives to blood transfusion on a case-by-case basis, weighing the potential risks and expected benefits for each patient.

The basic features of such a program should include (1) the participation of patients scheduled for elective procedures in preoperative autologous blood donation programs; (2) the discontinuation of aspirin therapy 1 week to 10 days before operation, if possible; (3) the continued use of standard intraoperative blood conservation techniques (hemodilution, intraoperative phlebotomy, reinfusion of oxygenator contents); (4) the expanded use of intraoperative blood processing (increasing use of Cell Saver to process blood aspirated from the operative field, concentration and washing of oxygenator contents after CPB, processing of blood withdrawn before CPB into PRP); (5) the reinfusion of postoperative mediastinal drainage according to published guidelines; (6) the acceptance of strict criteria for transfusion in patients in hemodynamically stable condition (hemoglobin < 80 g/L [8.0 g/dL] or hematocrit < 0.25); and (7) the selective use of available pharmacological agents to prevent or treat the causes of bleeding after CPB. Aprotinin appears to have the most clinical usefulness, but is not currently available in the United States. Desmopressin acetate cannot be recommended for routine use at this time. A new antifibrinolytic agent, tranexamic acid, deserves further study [70].

Future Directions

Increasing our understanding of the events that occur at the blood–synthetic surface interface will result in the development of truly hemocompatible materials for use in extracorporeal circuits. By decreasing the damage that occurs when blood is exposed to a synthetic surface, the need for blood transfusions to replace damaged blood components or treat coagulation defects will be decreased. The role of pharmacological agents will be expanded through the use of growth factors, such as erythropoietin, which stimulate production of red blood cells by the body [71]. For the patient undergoing preoperative autologous donation, administration of erythropoietin may increase erythropoiesis threefold to fourfold after 7 to 10 days [72]. In one study [73] in which patients were administered recombinant human erythropoietin or a placebo over a 3-week period, 98% of patients receiving erythropoietin were able to donate at least 4 units of blood, whereas only 70% of those receiving a placebo could donate that number. Administration of erythropoietin may also result in a more complete recovery of red cell mass by the patient both before and after operation. Finally, the development of synthetic blood substitutes promises to eliminate the need for homologous transfusions.

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Coarctation of the Aorta

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CORRESPONDENCE

Valve Insufficiency After Atrioventricular Septal Defect Repair: Differences Between Patients With and Without Down's Syndrome?

To the Editor:

In a recent issue of *The Annals* I read the interesting paper of Abbruzzese and associates [1] on the anatomical and surgical determinants of late mitral valve insufficiency after repair of partial atrioventricular canal. Unfortunately, Abbruzzese and associates did not report any data about the presence of Down's syndrome in their patients. How many of the 9 patients that underwent reoperation for mitral dysfunction had Down's syndrome? Was there a difference between children with and without Down's syndrome in their series? Because increasing attention is being paid to the anatomical [2, 3] and surgical [4, 5] differences between Down's and non-Down's populations with atrioventricular canal, I believe that these data must be analyzed and described even if not statistically significant.

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Reply

To the Editor:

The focus of our paper was on anatomical and surgical factors possibly associated with late left atrioventricular (AV) valve insufficiency after repair of partial AV septal defects. However, we are aware that Dr Marino's group is investigating the proposition that AV septal defects may be associated with a worse prognosis in children without chromosomal abnormalities, and we will gladly answer his questions.

None of the 9 patients who required reoperation in our series had Down's syndrome. Also, only 1 of the 16 patients (6.25%) in whom late left AV valve insufficiency developed had Down's syndrome. These figures show a lower incidence than that of Down's syndrome in our total series of partial AV septal defects (16 of 107 patients = 14.95%). These differences do not reach statistical significance ($p = 0.45$ and 0.58, respectively).

However, to correctly interpret these data, one must keep in mind that 15 patients, in whom the partial AV septal defect was part of a more complex malformation, were excluded from our study; none of them had Down's syndrome. Interestingly, similar to the findings of the group from the Ospedale Bambino Gesú [1], this series includes 3 patients with aortic coarctation and 2 patients with right ventricular dominance.

Moreover, when reviewing our total experience with reoperations for left AV valve repair failure in AV septal defects (all types) we noticed a very low incidence of Down's syndrome (2 of 22 = 9.1%) as compared with the 49% incidence of Down's syndrome in the total group of AV septal defects (all types). This difference is highly significant ($p < 0.001$).

A specific search for differences in the severity of this malformation in patients with and without Down's syndrome was not performed in our center as we believe that AV septal defects must be repaired in patients with and without Down's syndrome, regardless of their extent. More data are needed to confirm the controversial findings of Bull and associates [2] regarding an excellent life expectancy in complete AV septal defects with Down's syndrome without surgical repair.

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Left Ventricular Outflow Tract Obstruction With Aortic Arch Anomalies and Ventricular Septal Defect

To the Editor:

Iwahara and colleagues [1] have recently suggested that patients with a ventricular septal defect (VSD) and either interrupted aortic arch or coarctation, who have a left ventricular outflow tract diameter to ascending aortic diameter ratio of less than 0.6, should not undergo a standard reparative procedure of VSD closure and correction of the arch anomaly. They have based their conclusion on 4 patients with interrupted aortic arch and 13 patients with coarctation and VSD. Three of 4 patients with a ratio less than 0.6 died, whereas 2 of 13 patients with a ratio greater than 0.6 died. They conclude that the presence of severe left ventricular outflow tract obstruction (as determined by a left ventricular outflow tract to aortic ratio of less than 0.6) "clearly necessitates modification of present surgical strategies."

Our recent review [2] of 37 patients with interrupted aortic arch and VSD as the only associated anomaly has led us to an alternative conclusion regarding the clinical significance of left ventricular outflow tract obstruction. Only 2 of the 37 patients (5%) underwent modification of their initial procedure because of the presence of suspected severe subaortic stenosis. In 8 of the 37 patients, evidence of subaortic stenosis subsequently developed after repair. All cases became apparent within 3 years of repair and have been treated successfully by conventional methods. In

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spite of this conservative approach to the management of subaortic stenosis with interrupted aortic arch, there has been a dramatic improvement in survival after one-stage reparative operation, so that by 1986 the probability of death within 2 weeks of repair was 7%.

Retrospective echocardiographic analysis of the left ventricular outflow tract diameter did not reveal this variable to be a useful predictor of outcome in our series. Neonates with a left ventricular outflow tract diameter as small as 3 mm have done well, whereas in others with greater diameters left ventricular outflow tract obstruction has subsequently developed. We have speculated that VSD closure results in a shift in interventricular septal position resulting in an increase in the effective diameter of the left ventricular outflow tract, perhaps analagous to the situation with left ventricular outflow tract obstruction and transposition after an arterial switch procedure. One probable predictor of subsequent development of left ventricular outflow tract obstruction was found to be the presence of an aberrant right subclavian artery arising from the descending thoracic aorta in patients with type B interrupted aortic arch [3].

The arterial pH immediately before surgical repair was an important predictor of subsequent outcome in our series. We note that the mean pH of patients in the high mortality group from Iwahara's series was 7.26 with a base excess of −8.7. We believe that aggressive resuscitation of the neonate with interrupted aortic arch including not only the use of prostaglandin E_1 to maintain ductal patency but also ventilation with room air to improve systemic perfusion, appropriate use of inotropic agents and antibiotics, and probably most importantly, waiting an appropriate period of time so that all variables have normalized before proceeding to operation, are more likely to improve the outcome of operation rather than complex modifications of various palliative surgical options.

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Reply

To the Editor:

The reason why our variable did not fit into their series may be related to the incidence of posterior deviation of infundibular septum as a cause of left ventricular outflow tract obstruction. Sennari [1] reported the high incidence of posterior deviation of infundibular septum in Japanese patients with aortic arch anomaly and VSD, whereas Anderson and associates [2] reported a lower incidence of posterior deviation of infundibular septum in their series. In fact, all patients with malalignment VSD in our series had associated posterior deviation of infundibular septum. Because our variable was made based on this patient setting, we believe it fits the patients with posterior deviation of infundibular septum in the series of Sell and associates [3].

The arterial pH indicated in our paper was measured on admission, not immediately before surgical repair. Of course severe acidemia in the high-mortality group was corrected by administration of prostaglandin E_1, inotropic agents, diuretics, or mechanical ventilation until surgical repair. We also believe as they do that aggressive resuscitation before operation would improve the outcome of operation.

Generally, the mortality rate in patients with aortic arch anomaly and VSD is still high [4], although Sell and associates [3] showed good results and the possibility of one-stage operation to become the best choice for patients with aortic arch anomaly and VSD. One-stage operation may become the best choice as they recommended if reasonable risk can be demonstrated, but the optimal surgical management of severe left ventricular outflow tract obstruction in patients with this anomaly has not been generally agreed on. Some variable to define the severity of left ventricular outflow tract obstruction is still necessary to raise the survival rate.

We believe our variable can help in the management of this anomaly until the mortality rate of one-stage operation becomes low at most of the main centers in the world. At least one should consider the severity of left ventricular outflow tract obstruction before operation, whichever operation is chosen.

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Diaphragmatic Plication

To the Editor:

The recent paper by Graham and collaborators [1] clearly indicates the interest in diaphragmatic plication for unilateral diaphragmatic paralysis. It should be remembered that paralysis and so-called eventration have the same aspects and provoke the same physiological disturbances, and that it is difficult to distinguish one from the other, even at thoracotomy or on biopsies. This explains controversies on the subject.

In 20 years my colleagues and I operated on only 24 patients: 13 children and 11 adults. Seven children (aged 5 hours to 9 months) were admitted in respiratory distress and under ventilation; 4 were in chronic respiratory insufficiency. Eight adults had respiratory symptoms and 3 had digestive symptoms. In all patients but 1, plication without incision of the diaphragm was performed through a thoracotomy. In 1 case a Bochdalek's hernia had been misdiagnosed and the plication was done through a laparotomy. The operation is intended to decrease lung compression, to strengthen the action of intercostal and thoracic muscles by allowing more fixity to the thoracic base, and to stabilize the mediastinum.

Four children died who had been operated before the 10th day of life: 2 from an associated malformation, 1 from a hyaline membrane, and 1 (premature) from fibrinolysis. The 9 survivors have been followed up for a period of 5 months to 12½ years (mean, 6.6 years). All were asymptomatic, and the position of the plicated diaphragm was maintained.

The 11 adults have been followed up for a period of 3 months to 18 years (mean, 8.5 years). Nine were asymptomatic, in 1 the dyspnea had decreased, and in 1 coexisting reflux persisted and was surgically cured. Chest roentgenograms showed a normal aspect in 7 cases and four persisting—though smaller—malpositions of the diaphragm, which never were greater than one intercostal space in height.

In adults, preoperative respiratory tests showed a 21% mean decrease of vital capacity and a 25% mean decrease of forced expiratory volume in 1 second. The postoperative tests showed a mean amelioration of 20% of vital capacity and 15% of forced expiratory volume in 1 second.

During the same period we decided not to operate on 4 children (aged 4 months, 7 months, 7 years, and 13 years) who had few or no symptoms; they have stayed symptomless, but 1 has reflux. In no case did the diaphragm spontaneously improve its position. We also did not operate on 6 adults who had symptoms that did not appear correlated with the anomaly; they could not be checked.

In newborn children, the prognosis depends on associated lesions and the condition of the lung. The matter is to know whether the paralysis might be reversible in cases of respiratory insufficiency. We operated after a mean period of 17 days (range, 2 to 47 days). It seems sensible not to wait more than 2 weeks with the baby on a ventilator.

In older children and in adults, the problem is more simple: the plication is justified when the anomaly produces symptoms, whatever its cause, excluding malignancy. Symptoms may appear late in life, when the pulmonary reserve decreases, or they may be digestive.

We completely agree with Graham and colleagues that plication is simple, safe, and durable. Its benefit can be measured. We personally never saw an unplicated diaphragmatic paralysis or eventration resume a normal topography, though improvement is possible, but probably slow. Too long a delay might be dangerous, especially for children in respiratory distress.

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Choice of Surgical Clips for Mammary Artery Take-Down

To the Editor:

In their recent article, Gold and colleagues [1] presented experimental data suggesting that both titanium and tantalum surgical clips can be safely used in patients exposed to diagnostic magnetic resonance imaging. This is a timely and interesting article, as more cardiac surgeons are using titanium clips for mammary artery take-down, believing titanium is less likely than tantalum to become displaced during magnetic resonance imaging and cause subsequent injury to mediastinal structures. Although titanium microclips are considered radiopaque, they are hardly visible on posteroanterior and lateral chest roentgenograms. Tantalum clips, on the other hand, are easily seen on these views and thus offer a clear picture of the course of the mammary artery graft, enabling the surgeon to follow its relationship to the sternum on the sagittal and coronal planes. This is critical for a safe resternotomy in the increasing population of patients with patent internal mammary artery grafts requiring coronary artery bypass reoperations, especially if no special mammary artery views are available on the cardiac catheterization.

The literature has repeatedly confirmed the safety of performing magnetic resonance imaging in the presence of small metallic clips in the mediastinum [2, 3], and Gold and colleagues have concluded that tantalum clips can be used as safely as titanium. For this reason, and the advantages mentioned above, we strongly recommend the use of tantalum clips in mammary artery take-down.

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Reply

To the Editor:

My colleagues and I are grateful for the comments of Dr Hovaguimian and Dr Floten. I concur wholeheartedly that the radiographic imaging of patients before reoperation median sternotomy for revascularization of the coronary system or valvar procedures is important, particularly in the setting of prior internal mammary artery harvesting. Given the frequency of internal mammary artery use and the excellent long-term patency of the internal mammary artery graft, the recurrent problem of jeopardizing an internal mammary artery–dependent coronary circulation will clearly become more common. Use of surgical clips to control side branch bleeders as well as to identify the course of the internal mammary artery pedicle is facilitated by the radiopacity of the clips. Given their safety in postoperative imaging, use for these applications can certainly continue to be supported.

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Internal Mammary Artery-to-Pulmonary Artery Shunt in Infants

To the Editor:

Although the classic Blalock-Taussig shunts continue to play an important role in the management of cyanotic congenital malformations, the anatomy may not be suitable for direct subclavian-to-pulmonary artery anastomosis in some infants. A number of modifications of the Blalock-Taussig shunt have been described [1–3].

We attempted to use internal mammary artery-to-pulmonary artery shunts on the hypoperfused side of the pulmonary artery in 2 infants (aged 3 months) with pulmonary atresia and asymmetrical pulmonary perfusion due to severe stenosis of the pulmonary artery bifurcation.

The operation was performed on the right side (hypoperfused side) through an anterolateral thoracotomy in the fourth intercostal space. The pulmonary artery was dissected and encircled with a tape for the application of the vascular clamp. The preparation of the internal mammary artery was carried out from the thoracic inlet to the level of the fourth intercostal space. With some ligation of branches, the internal mammary artery pedicle was easily isolated to obtain sufficient length and mobility. Heparin (1 mg/kg) was given intravenously. Interrupted 6-0 or 7-0 Prolene (Ethicon, Somerville, NJ) sutures were used for end-to-side anastomosis of the internal mammary artery to the right pulmonary artery. Postoperative angiography showed good patency of the anastomosed internal mammary artery (Fig 1).

With the introduction of expanded polytetrafluoroethylene (ePTFE) as a satisfactory substitute, the modified Blalock-Taussig shunt using an ePTFE prosthesis has become increasingly popular as an acceptable alternative to the classic Blalock-Taussig shunt [2, 3]. In recent years, some reports indicated that the long-term success rate of the modified Blalock-Taussig shunt using ePTFE conduits 4 mm in diameter would be disappointing when the palliative procedure would be expected to last several years. Subsequently, based on the theory that the subclavian artery orifice might act as a flow regulator in prosthetic–pulmonary artery shunts, ePTFE grafts at least 5 mm in diameter were recommended for use even in small infants [2, 3].

If there is discontinuity of the right and left pulmonary arteries, the first shunt tends to be constructed on the hypoperfused pulmonary artery side, as the occlusion of the dominant pulmonary artery side during anastomosis should be avoided. When the bilateral systemic-to-pulmonary artery shunts are considered to be necessary in infancy, a larger graft has a potential risk of early death related to congestive heart failure due to an overflow of the lungs. Moreover, there are some associated disadvantages of modified Blalock-Taussig shunts including shunt infection and serous fluid leakage from the ePTFE shunt graft.

To the best of our knowledge, there are 2 reported cases of use of internal mammary artery 5 mm in diameter as a palliative systemic-to-pulmonary shunt in older cyanotic children [4, 5]. Cobanoglu and co-workers [6] used internal mammary artery as a preparatory shunt in infants with coarctation of aorta, ventricular septal defect, and absent right pulmonary artery. Probably because of the presence of coarctation of the aorta, the diameter of the internal mammary artery reached 3 mm in their 3-month-old infant.

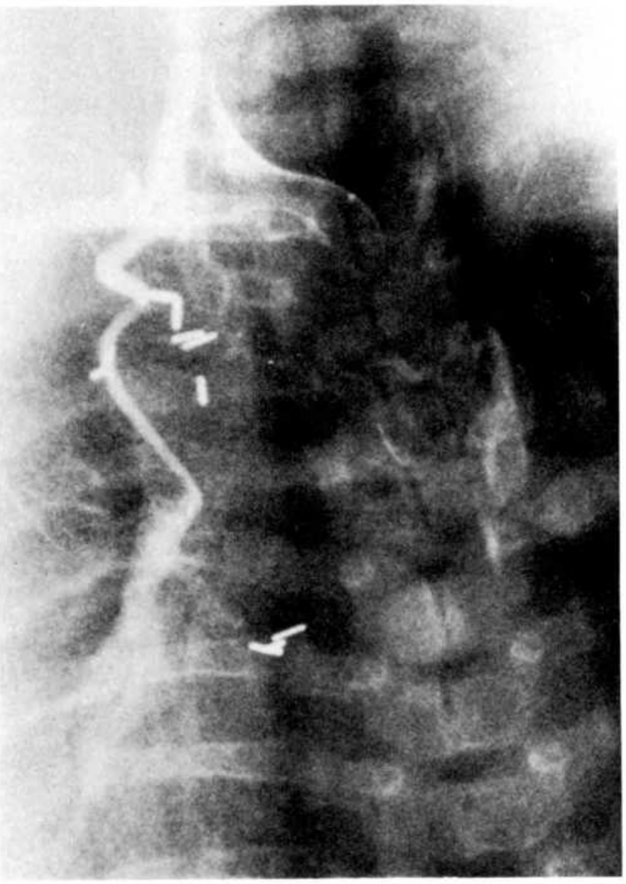

Fig 1. Postoperative angiogram of internal mammary artery-to-pulmonary artery shunt. Patency of shunt was demonstrated.

We report use of an internal mammary artery as a graft for initial systemic-to-pulmonary shunt in small infants with severe stenosis at the pulmonary artery junction and without coarctation of the aorta. This procedure was proposed to stimulate the development of the pulmonary artery on the hypoperfused side and to reduce the likelihood of excessive pulmonary flows when a bilateral shunt was performed in infants not solely as a means of obtaining a stable circulation and oxygenation during the construction of the dominant side shunt. With this operative technique, moreover, bilateral palliative systemic-to-pulmonary shunt with native artery can be constructed without the impairment of bilateral vertebral arterial circulation.

In conclusion, focusing on the usefulness of internal mammary artery as a palliative systemic-to-pulmonary artery shunt in small infants with severe stenosis at the bifurcation of the pulmonary artery, a longer follow-up is necessary for the validity of these shunts, but the early results are encouraging.

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Free Gastroepiploic Artery Graft

To the Editor:

We read with interest the article by Tanimoto and associates [1] on multiple free (aorta–coronary) gastroepiploic artery grafting. We have used the right gastroepiploic artery as a graft for coronary bypass and, to date, remain enthusiastic for its use in selective instances after an experience with more than 80 cases. However, we have a word of caution. In our report in this journal [2], we found that 3 of 10 patients with early postoperative angiography had areas of spasm in the free gastroepiploic grafts. Carter [3] of Australia, in a study of 30 patients, noted angina in the early postoperative period in patients with gastroepiploic artery grafts, yet the angina disappeared within 1 month. With pharmacological studies of this vessel we have noted that it is a very vasospastic artery, much more so than saphenous vein grafts and, preliminary data indicate, more so than internal mammary artery grafts. We, therefore, have discontinued its use as a free graft except in unusual circumstances and then attempt to graft it only to coronary arteries of lesser importance. We are awaiting long-term studies on our patients who have had free right gastroepiploic artery grafts before electively using this vessel as a free graft.

Tanimoto and associates [1] demonstrated a postoperative patent graft in Figure 2 in their report; however, they did not indicate the time at which the angiogram was taken in the postoperative period.

We congratulate Tanimoto and associates on their use of all arterial conduits and understand the reluctance to use the saphenous vein in their particular patient. However, we would offer a word of caution.

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Esophagectomy for Barrett's Esophagus

To the Editor:

I read the article by Altorki and colleagues [1] with interest. It does, however, raise questions related to the most difficult aspect of Barrett's esophagus: indications for operation or more conservative management in the setting of severe dysplasia. It is a dilemma for pathologists, internists, and surgeons alike.

Barrett's esophagus consists of metaplasia into epithelium of three basic types: fundic, gastric cardia, and specialized columnar. Recent studies have shown that of these three types, the specialized columnar is most likely to progress to adenocarcinoma [2, 3]. Further, it has been noted that both high-grade dysplasia and in situ carcinoma can occur in grossly intact mucosa, which makes initial endoscopy difficult and potentially misleading [4]. Altorki and associates did not describe the histological findings in the patients with high-grade dysplasias or those in whom esophagectomy was performed to "rule out cancer." It would be instructive to know the types of metaplastic epithelium seen, the rate of progression of dysplasia in those patients operated on, and the preoperative endoscopic findings. Also, 88 patients were referred over a 10-year period: how many of them had dysplasia of any grade when first seen, over what interval of time did dysplasia or carcinoma develop, and what is the outcome for the entire group?

Finally, there is another difficult situation not described here: multiple or extensive areas of severe dysplasia. Do such patients require early surgical intervention, even proceeding to operation without a trial of medication?

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Reply

To the Editor:

My colleagues and I certainly agree with Dr Weil that patients with Barrett's esophagus and associated high-grade dysplasia represent a clinical dilemma to surgeons, pathologists, and gastroenterologists alike. We grade high-grade dysplasia using the criteria established for inflammatory bowel disease [1]. High-grade dysplasia is accordingly defined as unequivocal neoplastic alteration of the metaplastic epithelium with cytological and architectural changes up to and including those seen in carcinoma in situ. The distinction between carcinoma in situ and high-grade dysplasia is often taxing even for experienced cytopathologists and from a practical standpoint is probably irrelevant. Three of our patients underwent resection for high-grade dysplasia. The metaplastic epithelium was of the intestinal variety in 1, junctional in 1, and a combination of all three types in 1 patient. We have since reported on a series of patients resected solely for high-grade dysplasia with invasive carcinoma detected in 40% of resected specimens [2]. All are alive and well on long-term follow-up.

Two patients underwent resection owing to our inability to definitively exclude a diagnosis of carcinoma preoperatively. One had a large obstructing exophytic lesion and the other had an undilatable stricture. The metaplastic epithelium was of the junctional type in 1 and of the intestinal type in the other.

Finally, the issue of multiple or extensive areas of high-grade dysplasia raised by Dr Weil has been addressed by careful pathological and flow cytometric studies in patients with associated Barrett's adenocarcinoma [3]. This clinical situation represents an epithelial surface that is genomically unstable and probably sets the stage for malignant transformation.

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Oxygenator Failure

To the Editor:

The recent pieces of correspondence regarding oxygenator failure [1–4] are interesting and worth further considerations. Kurusz and associates [1], in addressing Peirce's editorial [5], agree that a more readily available reporting mechanism should be developed for perfusion-related devices, specifically in the area of oxygenators. Additionally, correspondence by Robblee and associates [3] and Peirce [4] address the issue of standards for the conduct of cardiopulmonary bypass and, perhaps more importantly, the issues of safety standards for cardiopulmonary bypass and associated procedures. Quality assurance reporting in the perfusion area has not had a true focus, nor has anyone taken a leadership role—we feel there is a need to do so.

Our group has had a 20-year operating history with the use of safety checklists for cardiopulmonary bypass and associated procedures. The checklists, much like the safety checklists used by pilots, are detailed and specific to the operation of the heart-lung machine and the various components of the cardiopulmonary bypass circuit. Currently, we are performing 10% of the annual number of cardiopulmonary bypass procedures done in the country, and all of our procedures require completion of safety checklists before institution of cardiopulmonary bypass.

Additionally, we have communicated with the Food and Drug Administration and the Emergency Care Research Institute to pass along information regarding product concerns as well as guidelines for clinical procedure protocol development. We have developed, as part of our perfusion protocols, a series of redundant safety systems that are mandatory on all cardiopulmonary bypass circuits. In consulting with physicians nationwide, the concept of quality assurance reporting and safety standards has been very positively received.

A database is maintained that lists any variance that may occur during the procedure. Variances from clinical perfusion protocols, performance problems, or manufacturing defects of a product included in the cardiopulmonary bypass circuit are tracked.

Monthly quality assurance variance reports are generated and reviewed to specifically address trends in the variances reported. These reports highlight any increased incidence of product failure, product defect, and variances from clinical procedure protocols. On a regular basis, we report to all of the manufacturers any product failures or defects and receive reports in return with an explanation from the manufacturer.

PSICOR uses a two-member perfusion team, two perfusionists or a perfusionist and perfusion assistant. This measure provides an additional element of safety to the monitoring and operation of the cardiopulmonary bypass circuit and associated systems.

We strongly believe that stricter and more broad-based perfusion standards should exist not only in the area of safety but in the area of standard practice for the conduct of cardiopulmonary bypass. In the absence of national standards, we have provided a very strict set of standards for safety and clinical perfusion. Our incidence of perfusion-related accidents is considerably lower than reported in the literature, and we feel this is attributable to the very comprehensive safety standards, quality assurance checklists, and clinical protocols we have implemented.

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Descending Necrotizing Mediastinitis

To the Editor:

The recent article by Wheatley and associates [1] comprehensively reviews descending necrotizing mediastinitis. We would like to add an unusual case of descending necrotizing mediastinitis to the literature on this complex surgical problem.

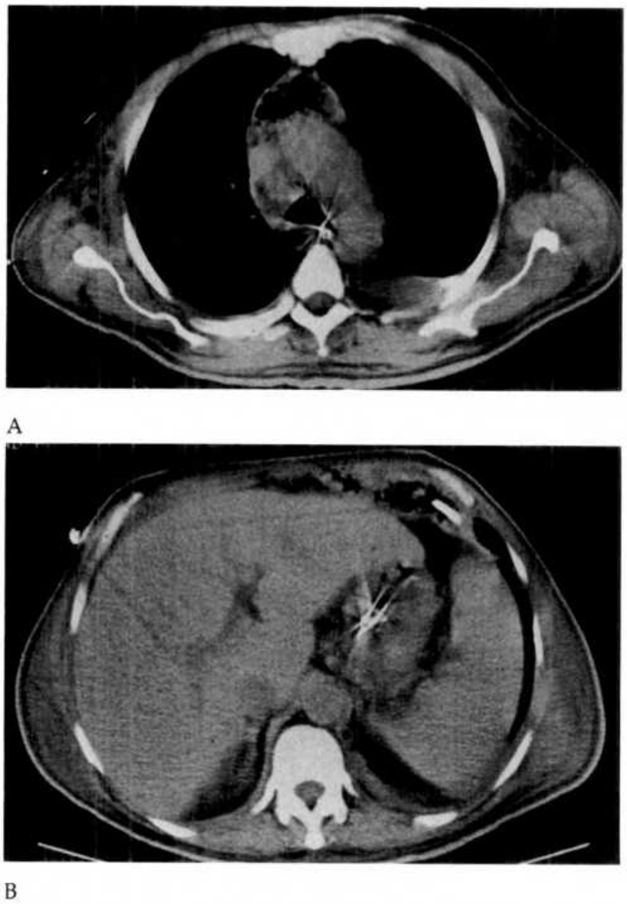

Fig 1. Computed tomographic scan of the chest shows a large mediastinal abscess. This gas-filled collection extended from the tracheostomy site to the epigastrium and then to the left retroperitoneum.

A 57-year-old diabetic man underwent craniotomy and evacuation of a subdural hematoma resulting from a fall. Ten days later, elective tracheostomy was performed at the bedside. Within the next 24 hours, the tube became displaced and had to be reinserted several times.

Over the next few days a wound infection developed at the tracheostomy site, and the patient's condition subsequently deteriorated with signs of systemic sepsis. A computed tomographic scan demonstrated a large mediastinal collection from the tracheostomy site to the epigastrium, extending into the left retroperitoneum (Fig 1). This infection was initially managed with a percutaneous drainage catheter inserted through the flank and directed into the retrosternal space under radiographic guidance.

The Thoracic Surgery Service was consulted because of persistent sepsis despite the catheter drainage and appropriate antibiotics. At operation, the retrosternal space was extensively drained from subxiphoid and cervical approaches, as depicted by Wheatley and co-workers [1]. The preperitoneal and retroperitoneal collections were drained through a left subcostal incision, and the tracheostomy wound was debrided. There was prompt resolution of sepsis; the drains were removed over the next few weeks.

Unfortunately, the patient experienced multiple problems and died 2 months after surgical drainage of the mediastinum. Autopsy determined the cause of death to be pneumonia and meningitis. There was no persistence or recurrence of the mediastinitis.

Mediastinitis is a rare complication of tracheostomy. In a review of 1,928 tracheostomies, there were only 2 cases of mediastinitis [2]. The cause of descending necrotizing mediastinitis in those cases was thought to be extension of a wound infection. In our patient, it is likely that initial problems with tube position resulted in wound infection as a consequence of injury and contamination of the peritracheal tissues. We would like to underline the importance of early, extensive drainage in patients with descending necrotizing mediastinitis.

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EVENTS OF INTEREST

Brief notices of meetings, courses, and symposia relevant to cardiothoracic surgery are published as a service. They should be formatted as follows: meeting title, date, place, sponsor, accreditation, director, fee, minimum/maximum attendance, and address/phone for further information. Announcements should be sent at least 4 months in advance of publication to *The Annals of Thoracic Surgery*, 3108 Queeny Tower, Barnes Hospital Plaza, St. Louis, MO 63110-1041; telephone, (314) 361-6084.

Thirty-seventh Annual Meeting of the Southern Thoracic Surgical Association, Cerromar Beach, Puerto Rico—November 8–10, 1990

For information on this meeting, contact Gordon F. Murray, MD, Southern Thoracic Surgical Association, 111 East Wacker Dr, Chicago, IL 60601-4301; or telephone (312) 644-6610 (facsimile: (312) 938-1214).

Sixty-third Scientific Session of the American Heart Association, Dallas, Texas—November 12–15, 1990

For information on this meeting, contact American Heart Association, 7320 Greenville Ave, Dallas, TX 75231; or telephone (214) 750-5300.

Global Plenum on Organ Transplantation, Lucknow, India—December 14–16, 1990

For information on this meeting, contact Prof Probal Ghosh, Plenum Secretariat, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Raebareli Rd, Post Box 375, Lucknow 226001, India.

Cardiopulmonary Rehabilitation: Status '91, Gainesville, Florida—January 28–30, 1991

For information on this meeting, telephone Michael L. Pollock, PhD, at (904) 392-9575.

Twenty-seventh Annual Meeting of The Society of Thoracic Surgeons, San Francisco, California—February 17–20, 1991

For information on this meeting, contact Richard P. Anderson, MD, The Society of Thoracic Surgeons, 111 East Wacker Dr, Chicago, IL 60601-4301; or telephone (312) 644-6610.

Fortieth Annual Meeting of the American College of Cardiology, Atlanta, Georgia—March 3–7, 1991

For information on this meeting, contact American College of Cardiology, 9111 Old Georgetown Rd, Bethesda, MD 20814; or telephone (800) 253-4636.

First International Symposium on Diseases of the Aorta, Tokyo, Japan—March 6–8, 1991

For information on this meeting, contact Secretariat of ISDA, c/o Japan Convention Services, Inc, Nippon Press Center Bldg, 2-2-1, Uchisaiwai-cho, Chiyoda-ku, Tokyo 100, Japan.

Seventy-first Annual Meeting of The American Association for Thoracic Surgery, Washington, DC—May 6–8, 1991

For information on this meeting, contact American Association for Thoracic Surgery, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Quality of Life After Open Heart Surgery, Antwerp, Belgium—May 16–18, 1991

This meeting is sponsored by The World Health Organization, the European Society of Cardiology, and the International Society and Federation of Cardiology. For information on this meeting, contact P. J. Walter, MD, FACS, Professor and Chairman of the Department of Cardiovascular Surgery, University Clinic of Antwerp, B 2520 Antwerp, Wilrijkstraat 10, Belgium; or telephone 00323.829.11.11 (facsimile: 00323.830.20.99).

Coronary Surgery: Clinical and Basic Perspectives, Sheffield, England—May 17, 1991

For information on this meeting, contact G. D. Angelini, Clinical Sciences Centre, Northern General Hospital, Herries Rd, Sheffield S5 7AU, England.

Fifth International Symposium on Cardiac Bioprostheses, Avignon, France—May 24–27, 1991

This meeting is sponsored by the Scientific Committee for the Advancement of Cardiac Bioprostheses. For information on this meeting, contact Beverly Farrier, ProMedica International, 620 Newport Center Dr, Suite 575, Newport Beach, CA 92660.

Ninth World Symposium on Cardiac Pacing and Electrophysiology, Washington, DC—May 28–31, 1991

This meeting will incorporate the Twelfth Annual Scientific Session of the North American Society of Pacing and Electrophysiology. For information on this meeting, contact IXth World Symposium Secretariat, c/o NASPE, 377J Elliot St, Newton Upper Falls, MA 02164; or telephone (617) 237-1866 (facsimile: (617) 431-1991).

The International Society for Cardiovascular Surgery/The Society for Vascular Surgery, Boston, Massachusetts—June 3–5, 1991

For information on this meeting, contact the Association offices, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Seventeenth Annual Meeting of The Western Thoracic Surgical Association, Seattle, Washington—June 26–29, 1991

For information on this meeting, contact The Western Thoracic Surgical Association, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Twentieth World Congress of The International Society for Cardiovascular Surgery, Amsterdam, the Netherlands—September 1–6, 1991

For information on this meeting, contact the Registrar: XX World Congress—ISCVS, International Travel Service, Inc, 104 Wilmot Rd, Suite 300, PO Box 825, Deerfield, IL 60015; or telephone (708) 940-2100 (facsimile: (708) 940-2386); or contact the Congress Secretariat, International Society for Cardiovascular Surgery, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

CLASSIFIED ADS

Classified ads for positions wanted or available must relate only to the thoracic or cardiovascular surgical specialties. Display space (minimum, ¼ page) is also available through Media for Medicine, Inc, 440 Park Avenue South, New York, NY 10016 (telephone: (212) 684-5540; facsimile: (212) 545-0651). All ads are subject to the approval of the editor, and material deemed inappropriate for publication in *The Annals* will be rejected. If anonymity is desired, please indicate this at the time of submission and a code number will be assigned. All responses received will remain confidential in the publisher's office and will be conveyed to the advertiser shortly after receipt. The charge for all classified advertising is **$1.25 per word per insertion, minimum 20 words.** Abbreviations, dates, initials, post office box numbers, telephone numbers, years, and zip codes are considered one word each. **There is an additional fee of $17.00 per insertion for box number ads.** The copy deadline is 7 weeks prior to publication, eg, for the March issue, copy should be received by the 1st of January. Ad orders should be for a maximum of 6 months; ads may be renewed for up to 6 months at a time. Ad copy should be typed double-spaced and mailed in duplicate to: *The Annals of Thoracic Surgery*, Desk Editorial, Classified Ads, Elsevier Science Publishing Co, Inc, 655 Avenue of the Americas, New York, NY 10010 (facsimile: (212) 633-3853). Make *nonrefundable* check payable to Elsevier Science Publishing Co, Inc.

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SITUATIONS AVAILABLE

Cardiovascular and thoracic surgeon, board eligible/certified, wanted to join well-established private practice located in Ft. Lauderdale, FL. Send CV.

Please respond to Box 251. 251L/K

Cardiovascular and thoracic surgeon, board eligible or certified, wanted to join another surgeon in private practice in the Southeast. Please respond with curriculum vitae.

Please respond to Box 252. 252G/L

Cardiovascular and thoracic surgeon: Excellent opportunity to join a busy north central Florida group. Practice specializes in adult cardiac and thoracic surgery. Excellent benefit package. Please reply with curriculum vitae.

Please respond to Box 348. 348F/K

Cardiovascular and thoracic surgeon, BE/BC, to join in 2-man private practice in cardiovascular and thoracic surgery in the Northeast. Excellent benefits package. Association will lead to partnership. Please send curriculum vitae.

Please reply to Box 350. 350F/K

Cardiothoracic surgeon needed for established program in scenic Montana. Unique opportunity for congenial private practice lifestyle combined with high-tech hospital environment including dedicated cardiac OR, cardiac anesthesia, and cardiac intensive care unit. Solo practice or association leading to partnership. Could be experienced surgeon or recent graduate from quality program.

Please respond to Box 354. 354G/L

Thoracic surgeon wanted: A busy thoracic practice 20 miles south of Boston seeks to add a third surgeon by July 1991. Candidate must be board certified or eligible. This active practice operates from a multihospital referral base. The community is an ideal area for family living, with excellent schools and nearby Boston and Cape Cod cultural and recreational facilities. Competitive salary, malpractice insurance, and excellent benefit package. Please respond with CV.

Please respond to Box 356. 356G/L

Cardiothoracic surgeon, BE/BC, to join established 1-man private practice involved with adult cardiac, thoracic, and vascular surgery in major Midwest community. New or recent graduate preferred. Salary leading to partnership. Please send CV, clinical experience, and references.

Please respond to Box 366. 366H/K

Vascular surgeon, board certified/board eligible, recently trained, wanted to join a busy solo vascular surgeon in north central West Virginia using 300-bed very progressive hospital. Excellent package and potential.

Please respond to Box 367. 367H/K

Cardiac surgeon, board certified or board eligible, for very busy private practice group doing primarily adult cardiac and vascular surgery. Seeking an Associate to join well-established practice in northeast Ohio metropolitan area. Please respond with curriculum vitae and references.

Please respond to Box 370. 370H/A

Cardiothoracic surgery group seeking an additional board eligible/board certified surgeon with interests and skills in general thoracic surgery for busy expanding practice. Private 3-man group practices in the Southeast in university-affiliated regional medical center. Practice currently includes mainly adult and pediatric cardiac and pulmonary surgery. Excellent opportunity for suitable applicant.

Please respond to Box 384. 384H/K

Cardiothoracic surgeon, ABTS certified/eligible, with primary interest in cardiac surgery, wanted immediately to replace member of established surgical group practicing in Allentown, PA. Competitive salary/fringe benefits. Position leads to partnership for successful candidate.

Please send curriculum vitae, clinical experience, and references to Joan M. Persa, Administrator, 1275 South Cedarcrest Blvd, Allentown, PA 18103; (215) 821-8790. 393I/B

Cardiovascular and thoracic surgeon: BE/BC wanted to join well-established private group practice in Philadelphia area. Seek recent or upcoming graduate. Please send curriculum vitae.

Please respond to Box 394. 394I/B

Thoracic, vascular, and general surgeon to join a busy, all boarded, private practice on eastern Long Island. Should be board certified or eligible. Excellent salary and benefits, leading to a full partnership. Send curriculum vitae.

Please respond to Box 395. 395I/L

Cardiovascular surgeon to become second surgeon in new (two year old) adult cardiac program on west coast of Florida.

Submit CV to Manatee Cardiac Surgeons, PA, 201 4th Ave East #2, Bradenton, FL 34208. 396I/K

Cardiovascular surgeon, board certified/eligible, wanted to join two surgeons in an established cardiovascular program in the southeastern USA. Must have experience in adult cardiac, thoracic, and vascular surgical procedures. Salary/full partnership negotiable. Please send resume.

Please respond to Box 398. 398I/B

Cardiovascular and thoracic surgeon: Excellent opportunity for a board certified/eligible cardiovascular and thoracic surgeon to join in a new, growing department in a metropolitan New York community on Long Island. Academic/private practice setting with excellent compensation. 533-bed community teaching hospital. Experience in electrophysiologic surgery, artificial devices, and/or transplantation helpful. Please respond with curriculum vitae.

Please respond to Box 403. 403I/L

Thoracic/vascular surgeon needed to join prominent, active 3-man group in desirable suburban community in greater NY metropolitan area. Excellent opportunity for well-trained, highly motivated individual. Recent graduate preferred. Possible academic affiliation. Please respond with CV.

Please respond to Box 404. 404J/C

Congenital cardiothoracic surgery: The Children's Hospital of New Jersey is seeking a pediatric cardiothoracic surgeon to join its well-established program. Candidates should be board certified by the American Board of Thoracic Surgery and have 2 to 3 years experience in congenital cardiac surgery at fellowship and/or practice level. We are looking for a candidate with excellent surgical skills and a strong interest in research and teaching. The position carries an academic appointment at The University of Medicine and Dentistry of New Jersey–The New Jersey Medical School, which has an accredited program in Thoracic Surgery.

Send CV to Joseph V. Cotroneo, MD, The Children's Hospital of New Jersey/United Hospitals Medical Center, 15 South Ninth St, Newark, NJ 07107; (201)268-8671 (8606). 405J/K

Adult cardiac, peripheral vascular, and thoracic surgery practice in Midwest with rapidly growing referral base desires well-qualified surgeon for association leading to partnership. Practice includes busy transplant program. Academic affiliation available. Send CV and three references.

Please respond to Box 407. 407J/L

Thoracic and cardiovascular surgeon, board certified or eligible, wanted for an established practice in the Midwest with experience in adult cardiac, thoracic, and peripheral vascular surgery. Experience or training in heart/lung transplantation preferred. Initial contact should include curriculum vitae, clinical experience, and names of references.

Please respond to Box 408. 408J/B

Cardiovascular and thoracic surgeon—board eligible or qualified—wanted to join 4 physicians in very active private practice in greater Chicago area. Respond with curriculum vitae.

Please respond to Box 409. 409J/K

Vacancy exists for a BE/BC thoracic and cardiovascular surgeon. Candidate must have proven clinical and laboratory abilities and vascular surgery fellowship. Training required in major complex vascular procedures.

Please forward curriculum vitae to Carolyn G. Gray, 2002 Holcombe Blvd, Houston, TX 77030; or telephone (713) 795-7463. EOE. 410J/K

Assistant surgeon—Busy adult and pediatric cardiac and vascular surgery group in suburban Washington, DC, is seeking a non-board certified surgeon, preferably with cardiovascular experience, to assist in the operating room. Excellent salary and fringe benefits.

Please respond with CV to Virginia Heart Surgery Associates, 3301 Woodburn Rd, Suite 301, Annandale, VA 22003. 413J/L

Cardiac surgeon—board certified/eligible to be third member of established practice in suburb of Washington, DC, expanding to second hospital. Excellent compensation and benefits. Great opportunity for recent graduate wishing to locate in area of nation's capital. Practice focus is primarily in adult cardiac surgery with additional work in thoracic and peripheral vascular surgery available.

Please send curriculum vitae to PO Box 34945, Bethesda, MD 20817. 415J/C

Cardiovascular surgeon (BC/BE) to join 2 recently trained, board certified associates in a well-established adult cardiac surgery program in a multispecialty clinic affiliated with a stable referral system of 24 satellite clinics. Active program includes arrhythmia surgery. Midwest location is 30 minutes from the Minnesota lakes district.

Contact Dakota Medical Center, 1702 South University Dr, Fargo, ND 58103; Dr John Hicks, Director, Recruiting Division, (701) 280-3338. 416J/C

Cardiothoracic/vascular surgeon: board certified in general and thoracic surgery. Minimum of 2 years of practice experience. Very active private 5-surgeon multispecialty practice. Located in western Pennsylvania. Salary leading to full partnership. Excellent benefits. Detailed CV must accompany response. All replies treated confidentially.

Please respond to Box 419. 419J/C

Cardiothoracic surgeon—to join a 2-man private practice in cardiac, thoracic, and vascular surgery in the Great Lakes region. Must be board certified or eligible. Recent graduates considered. Send curriculum vitae.

Please respond to Box 421. 421J/C

Cardiothoracic surgeon needed to join established 2-man group in practice of adult cardiac, pulmonary, and vascular surgery. Must be board certified or eligible and graduate from a US medical school.

Please respond to R. W. Ruess, MD, 1834 Broadway, Cape Girardeau, MO 63701. 422J/C

Cardiovascular-thoracic surgeon—BE/BC to join established and growing 2-surgeon group in northeastern US. Practice includes high volume cardiac and general thoracic surgery. Recent trainee preferred. Submit CV.

Please reply to Box 423. 423J/C

Cardiothoracic surgeon wanted for busy, expanding private practice in Connecticut. Practice includes cardiac and thoracic surgery. Surgeon must be board eligible or certified. Excellent opportunity for suitable applicant.

Please respond to Box 424. 424K/A

Pacific Northwest opportunity for cardiothoracic surgeon with 2 to 4 years of experience. Growing multispecialty group practice in Portland, Oregon, seeks dynamic surgeon to join 3 other surgeons in our practice. General thoracic and vascular experience necessary.

Please send curriculum vitae to Dr James W. Asaph, MD, 507 NE 47th Ave, Portland, OR 97213-2282. 426K/D

Cardiothoracic surgeon, recent graduate, experienced in cardiac arrhythmia surgery, to join well-established cardiac surgical group in central Florida. Send CV.

Please respond to Box 427. 427K/A

Cardiac surgeon wanted to join established private practice in upstate New York, primarily adult cardiac. Board certified or eligible required. New or recent graduate preferred. Please send CV and references.

Please respond to M. A. Marvasti, MD, 101 Union Ave, #813, Syracuse, NY 13203. 429K/D

Faculty position: The Division of Thoracic and Cardiovascular Surgery, Southwestern Medical School of the University of Texas Southwestern Medical Center, is seeking an individual with strong clinical background in adult cardiovascular/thoracic surgery along with a commitment to education and research, for a position at the assistant professor level. Must be ABTS certified or eligible.

Send complete curriculum vitae to W. Steves Ring, MD, Chairman, Division of Thoracic and Cardiovascular Surgery, UT Southwestern Medical Center, 5323 Harry Hines Blvd, Dallas, TX 75235-8879. An equal opportunity employer. 430K

Wanted: BE/BC general surgeon with interest and experience in thoracic surgery who enjoys general surgery, or non-cardiac thoracic surgery, to assume practice of retiring double-boarded thoracic/general surgeon. 34-member multispecialty group in dynamic area hub city of 35,000, with six satellite offices in strategic locations and referral area of 300,000 plus. Superb hospital facilities, including CT, MRI, cardiac surgery, neurosurgery, etc. Directed towards those concerned about quality family lifestyle and professional concerns.

Contact Recruitment Coordinator, Park Clinic, 890 North Eisenhower Ave, Mason City, IA 50401; or dial 800-798-4321 for more information. 431K/D

Wanted: BC/BE cardiac surgeon to join an active cardiac surgical group in the Midwest. All aspects of adult and pediatric cardiac surgery performed along with an active artificial heart and transplant program.

Please respond to Box 434. 434K/L

Cardiovascular/vascular surgeon wanted to join busy established adult practice in Ohio. Recent graduate preferred. Please submit curriculum vitae. Association will lead to partnership.

Please respond to Box 436. 436K/C

BC/BE adult cardiothoracic surgeon wanted to join active established department in large multispecialty group practice located in metropolitan Midwest community. Medical school affiliation available. Respond with curriculum vitae.

Please respond to Box 437. 437K/D

SITUATIONS WANTED

Cardiovascular and thoracic surgeon, 45, ABS, ABTS, NE university trained, seeks relocation. Experienced, publications, academic appointment.

Please respond to Box 386. 386I/K

Cardiovascular and thoracic surgeon, university trained, 39, hard working, proficient in valve repairs, IMA grafting, redo surgery, experienced, wishes to relocate. Also available, cardiovascular anesthetist or intensivist to make self-sufficient team to start new practice. All inquiries handled with strictist confidence.

Please respond to Box 428. 428K/A

Cardiovascular and thoracic surgeon, 40, ABTS certified, university trained, experienced in establishing new program, seeks solo or group practice opportunities.

Please respond to Box 435. 435K/L

FELLOWSHIPS

Fellowship in cardiovascular surgery for 1 year available January 1 or July 1 with group doing approximately 1,000 cardiac procedures per year and large volume of vascular procedures. ABS eligible or certified, California medical license required or readily obtainable. Ideal for a person between general surgery residency and thoracic surgery residency.

Please respond with curriculum vitae to Bradley J. Harlan, MD, 5301 F Street, Suite 312, Sacramento, CA 95819. 364G/L

Cardiac transplant/device fellowship: Position available at The University of Arizona Health Sciences Center for a 1-year clinical fellowship in intrathoracic transplantation and circulatory device implantation beginning July 1, 1991. Applicants must be ABTS eligible/certified or foreign national physicians must have passed the FMGEMS and hold a valid ECFMG certificate.

Submit letter of application, curriculum vitae, and three letters of recommendation prior to January 1, 1991, to Jack Copeland, MD, Department of Surgery, University of Arizona, 1501 N Campbell Ave, Tucson, AZ 85724. 425K

Surgical residents/fellows—Clinical residency/fellowship positions available in adult and pediatric cardiothoracic surgery. Large volume of cases. New Jersey license required. Excellent salary and benefits.

Please contact Lynn B. McGrath, MD, Chairman, Department of Surgery, Deborah Heart & Lung Center, 200 Trenton Rd, Browns Mills, NJ 08015; (609) 893-6611, Ext. 359. 432K

Fellowship in vascular surgery—The Deborah Heart & Lung Center is currently seeking qualified applicants for the position of a Vascular Fellow beginning January 1, 1991.

Please contact Douglas H. Joyce, DO, Director, Surgical Residency/Fellowship Programs, 200 Trenton Rd, Browns Mills, NJ 08015; (609) 893-6611, Ext. 359. 433K

ACADEMIC CARDIOTHORACIC SURGEON

The Division of Cardiothoracic Surgery at Albany Medical College is increasing its fulltime academic faculty. Candidates should have interest in developing an active practice, participating in thoracic and general surgical resident training, as well as conducting clinical and laboratory research. Additional training or certification in areas such as critical care medicine, congenital cardiac surgery or electrophysiologic surgery is preferable. Albany Medical Center is an equal opportunity employer.

Please respond with curriculum vitae and letters of reference to Victor A. Ferraris, M.D., Ph.D., Head, Division of Cardiothoracic Surgery, Albany Medical College/ A-61, Albany, New York 12208.

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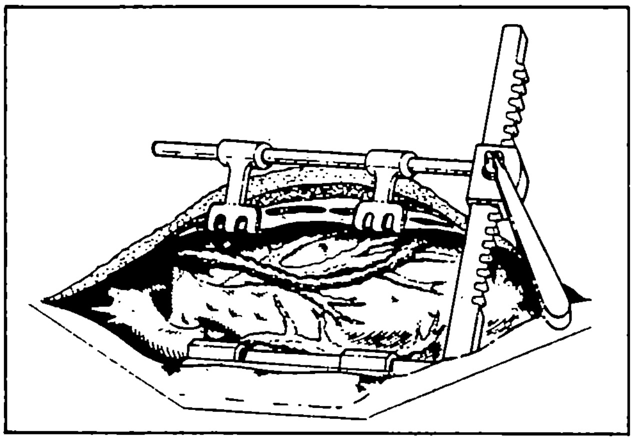

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West Virginia University (WVU) Health Sciences Center, Department of Anesthesiology, is recruiting for a pediatric cardiac anesthesiologist. Our Department currently has 15 faculty members and sponsors an approved residency training program, which has 24 residents.

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WVU is an Equal Opportunity/Affirmative Action Institution.

For additional information, contact:

**Edward Stullken, M.D., Chairman
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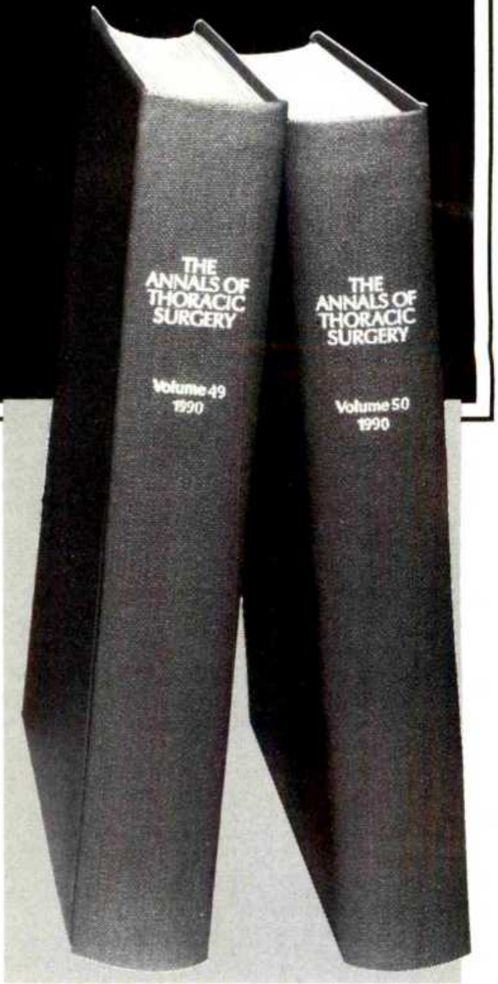
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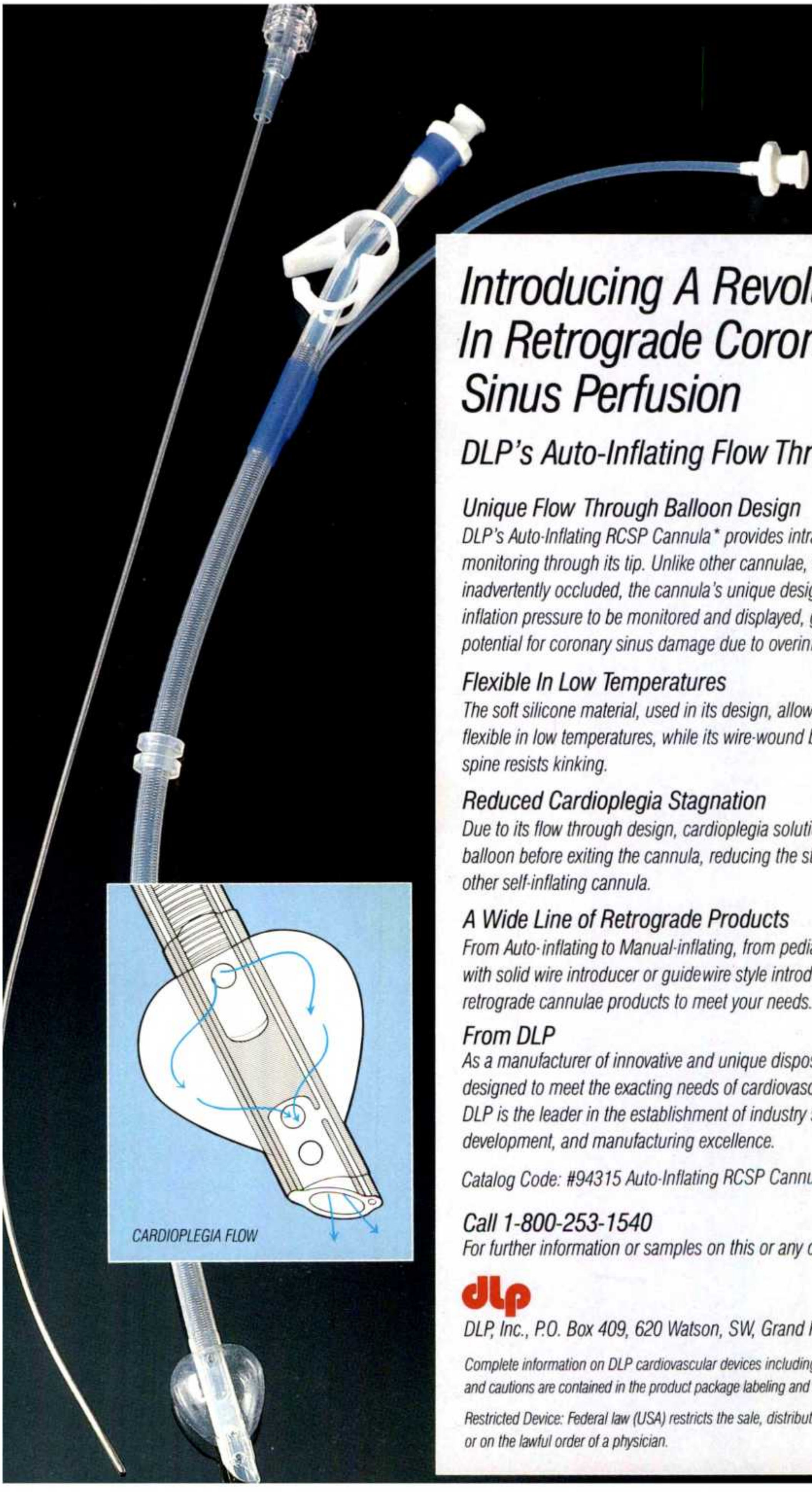

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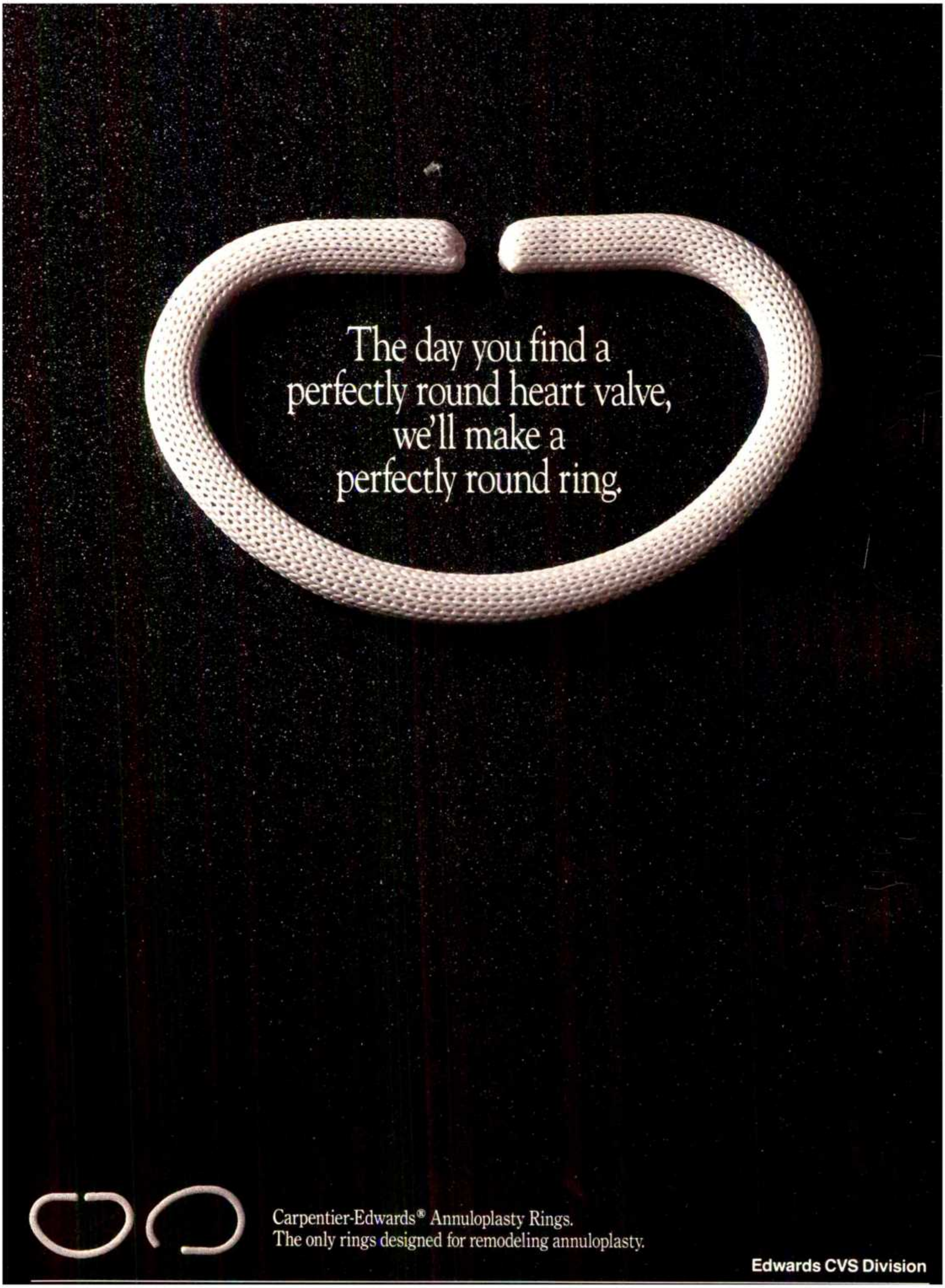

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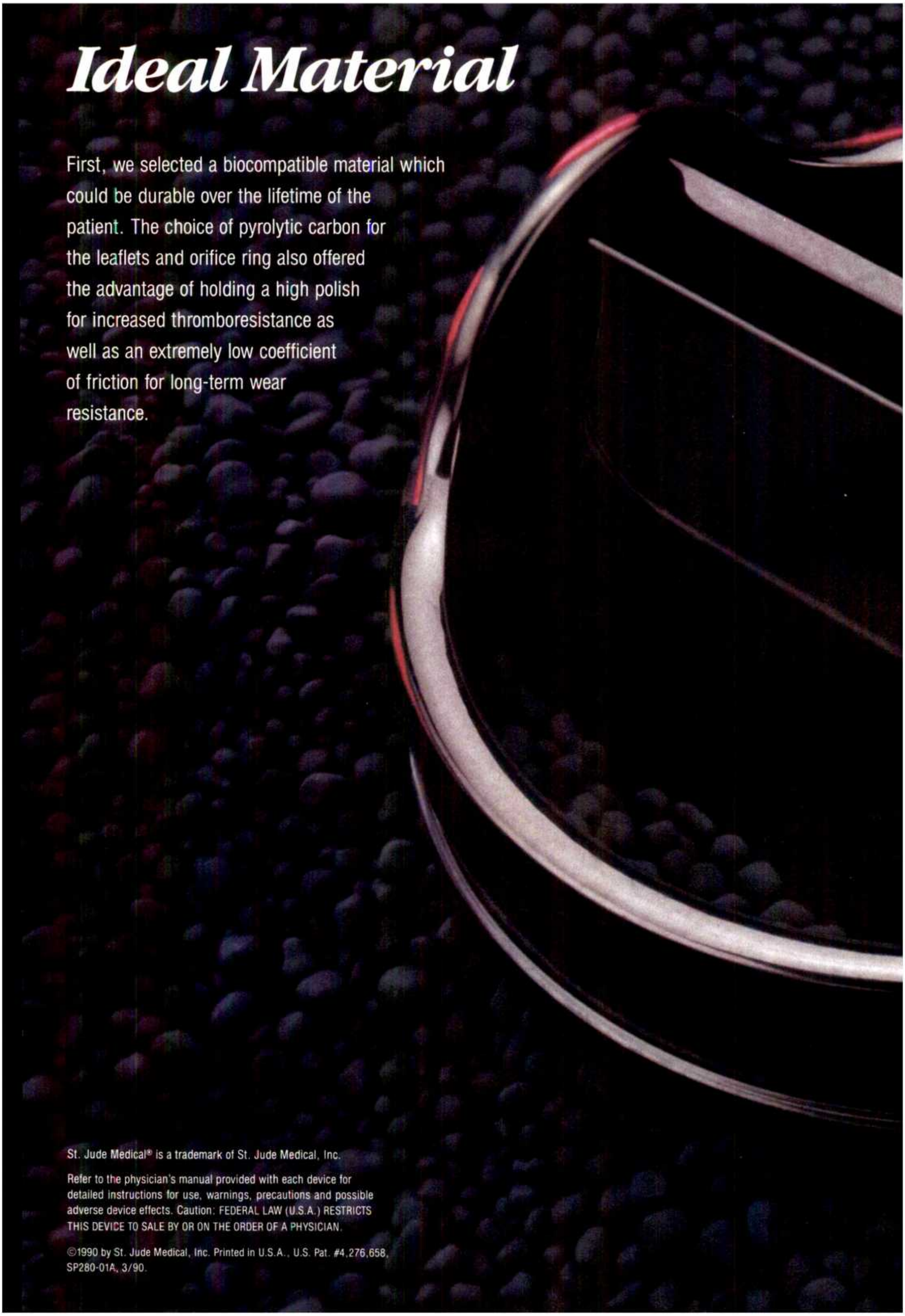

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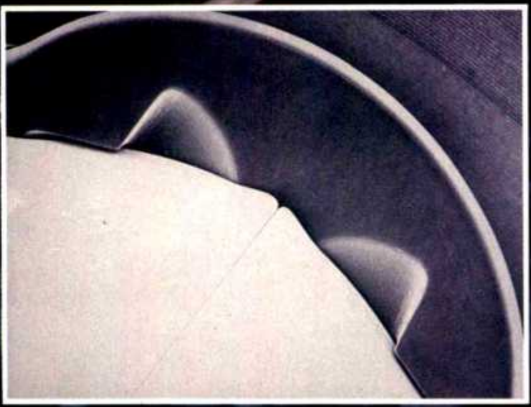

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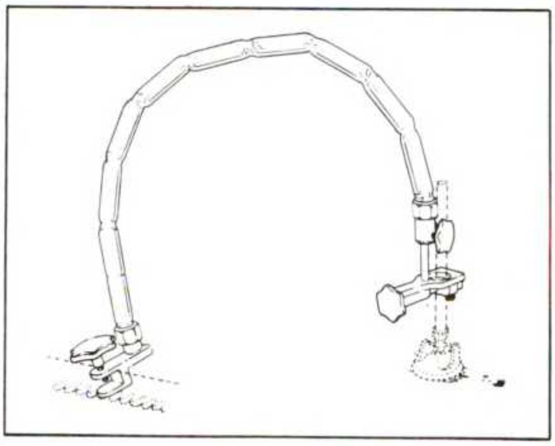

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Editorial Board Changes

Dr Clarence S. Weldon has completed two terms of service on the Editorial Board. He was a valued member of the Board for 6 years. The Editor and readers of *The Annals* are grateful to him for his dedication and expertise. He has received a certificate of recognition from The Society.

The Council of The Society has approved the appointment of Dr Clement A. Hiebert, Visiting Professor of Surgery, Toronto General Hospital, Toronto, Ontario, Canada, who began service on the Editorial Board in November 1990.

Clarence S. Weldon

Clement A. Hiebert

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Epidural Analgesia: Different Strokes for Different Folks

L. Penfield Faber, MD

Department of Cardiovascular and Thoracic Surgery, Rush Medical College, Chicago, Illinois

The effective control of pain after a thoracotomy not only provides patient comfort but also decreases the incidence of postoperative complications. Minimal postoperative pain allows the patient to breath deeply and cough forcefully. The clearing of retained secretions permits full expansion of the residual lung to minimize postoperative space problems with a resultant decrease in the incidence of prolonged parenchymal air leak. An effective cough avoids atelectasis and premature airway closure with its associated shunting and hypoxemia.

In this issue Brodsky and associates [1] describe their experience with the continuous lumbar epidural infusion of hydromorphone to treat postthoracotomy pain. This series of 44 patients demonstrates that a continuous infusion of epidural hydromorphone provides adequate pain relief. My colleagues and I [2] reported our early results in 1984, demonstrating the benefit of continuous epidural infusion of morphine for pain relief after thoracotomy when compared with intermittent epidural injections of bupivacaine or intermittent epidural injections of morphine.

See also page 888.

Brodsky's series does demonstrate good pain relief, and we also have obtained good pain relief after all types of surgical procedures; differences in technique and drug selection warrant discussion.

Respiratory depression associated with epidural narcotics can be a serious and even life-threatening complication after pulmonary resection. Whatever drug regimen is used for epidural analgesia, the avoidance of respiratory depression should play a major factor in the selection of which drug regimen is administered. Brodsky and associates noted that 6 of 44 patients suffered respiratory depression and required treatment with naloxone. This incidence of 13.6% would appear to be a rather high incidence of this complication and would suggest that drug dosage should be modified or possibly even that the drug should be changed. Experience at the Rush-Presbyterian-St. Luke's Medical Center with epidural analgesia in more than 1,000 thoracotomies has led us to our current drug regimen of fentanyl or morphine in combination with bupivacaine given as a continuous infusion. During the past 3 years, we have maintained a database of 504 thoracotomy patients having postoperative and epidural analgesia and have closely monitored the adequacy of analgesia and all associated morbidity. Our current techniques have been refined based on the problems we have encountered over the past 7 years and we currently continuously infuse either a fentanyl (0.001%)–bupivacaine (0.1%) or morphine (0.01%)–bupivacaine (0.1%) mixture through an epidural catheter placed in the region of the thoracotomy incision. The most recent evaluation of these data indicates that epidural fentanyl-bupivacaine infusions have provided analgesia similar to the morphine-containing solutions with fewer side effects [3]. Brodsky and associates cite the literature to indicate that there is a higher incidence of sedation, pruritus, and respiratory depression with fentanyl. This has not been our experience (Table 1).

In our group of 504 patients, there was an incidence of respiratory depression of 0.6% (3/504). An additional point to consider when choosing an epidural drug regimen is to be aware of the synergy between local anesthetics and narcotics. Synergy between epidurally administered local anesthetics and narcotics have been demonstrated in our clinical studies [4, 5]. It is our opinion that this combination produces better pain relief than with either drug alone.

Brodsky and associates recommend an intraoperative bolus infusion of hydromorphone 1 hour before completion of the procedure. One might ask why continuous infusion is not given intraoperatively as it is the preferred technique postoperatively. We begin the continuous epidural infusion shortly after induction of the anesthetic and it is maintained throughout the procedure. This technique allows the patients to awaken with good analgesia. Beginning a continuous infusion early rather than giving a bolus 1 hour before thoracotomy closure might decrease the rather high incidence (68%) of inadequate analgesia during the day of operation, which necessitates repetitive boluses of epidural fentanyl for supplemental analgesia [1].

Brodsky and associates describe their practice of a combined epidural and light general anesthetic using 16 to 22 mL of 2% lidocaine or 0.5% bupivacaine. A possible problem associated with this technique is the potential for hypotension from a sympathetic block. We also have had experience with this technique and have found our current continuous infusion of the described drug regimen to allow for a smoother intraoperative course.

A final point of variance is the placement of the epidural catheter at the lumbar level. Numerous studies have demonstrated that epidural morphine produces good pain relief after thoracotomy when administered through a catheter placed at the lumbar level. This is due to the

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Table 1. Postoperative Epidural Pain Management in 504 Thoracic Surgery Patients

Variable	Day 1	Day 2	Day 3
Visual analog pain scores	2.13 ± 2.5	1.39 ± 1.9	0.82 ± 1.4
Supplemental narcotic (morphine equivalents)	3.6 ± 8.1	3.5 ± 6.2	2.2 ± 4.9
Respiratory depression (respiratory rate < 10 bpm)	0.6%	. . .	. . .
Sedation	2.0%	. . .	. . .
Pruritis	14.9%	. . .	. . .
Nausea/vomiting	11.9%	. . .	. . .

hydrophilicity of morphine, which permits it to partition itself in cerebral spinal fluid and cover large segments of the spinal cord. Lipophilic agents, such as fentanyl, produce a more segmental effect and are more effective when administered through an epidural catheter placed at the site of the incision. Hydromorphone is more hydrophilic, having a lipid partition coefficient 8 times greater than morphine, whereas fentanyl is approximately 570 times more hydrophilic than morphine. Therefore, it is expected that the late respiratory depression effects of hydromorphone would be similar to those of morphine. Fortunately, early or late apnea has not been noted in Brodsky's series.

It may be different strokes for different folks, but the end result is the same: continuous epidural infusion after thoracotomy provides good pain control. I personally recommend it highly. The advantages of epidural analgesia outweigh its risks or side effects, but it is not innocuous therapy that can be delivered in a cavalier fashion. In any setting where epidural analgesia is used, there must be an anesthesiology staff who are expert in epidural catheterization and are committed to providing the extra time needed for postoperative care. This includes conducting in-service training for the hospital nursing staff, writing policy and procedure protocols that detail the specifics regarding patient management, and ensuring that patients are seen on a daily basis, or more frequently as needed, by an anesthesia care team member. In addition, communication between the surgical and nursing staff is essential to ensure patient safety.

Initiating a postoperative analgesic service may initially sound somewhat simple, but it can be a formidable task. A recent chapter describing the organization of a postoperative pain service outlines the important factors that must be addressed [6].

It is important to be aware that the technique of epidural analgesia can improve patient care and outcome while reducing hospital costs, but it does carry with it certain risks that require close surveillance. Epidural analgesia can produce good pain control but does require an extra measure of vigilance and postoperative care. It is best practiced when an experienced anesthesiology team maintains careful patient follow-up and communication with both the surgical and nursing staff as outlined by the Stanford group.

Special thanks to Dr Timothy R. Lubenow, Assistant Professor of Anesthesiology, Rush Medical College, for his assistance in the preparation of this editorial.

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Cardiac Transplantation for Neonates With Hypoplastic Left Heart Syndrome

John E. Mayer, Jr, MD

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In this issue of *The Annals*, Backer and co-workers [1] from the Children's Memorial Hospital in Chicago report their technique for orthotopic cardiac transplantation for neonates with hypoplastic left heart syndrome (HLHS). The technique involves the use of deep hypothermic cardiopulmonary bypass with bicaval cannulation and a period of circulatory arrest during which the donor aortic arch is used to reconstruct the recipient aortic arch. They report 4 patients treated with this technique with no operative mortality, and they found that the "hospital" ischemic time for the donor heart averaged 60 minutes. The average circulatory arrest time was 30 minutes. Backer and co-workers conclude that this technique offers the advantages of reduced donor hospital ischemic time so that the longer donor "travel" ischemic times could be accepted, and that the shorter circulatory arrest times should provide reduced neurological morbidity.

This report is certainly of interest to those involved in pediatric cardiac transplantation, although, as the authors have pointed out, it is an adaptation of the technique described by Cabrol. Backer and co-workers are to be congratulated on their results with this technique. As I read the manuscript, however, a number of issues came to mind. There seems to be little doubt that technical modifications that shorten the ischemic times of both donor heart and recipient brain are likely to improve outcome. What is less clear are the shapes of the curves describing the relationships between the incidence of unfavorable outcome and the ischemic time. Does the 20 minutes of cardiac ischemia saved by doing the right atrial and pulmonary artery anastomoses after removing the cross-clamp have a major impact on cardiac recovery if it represents 10% of the total ischemia time? Similarly, does an increase in cerebral ischemic time from 30 to 55 minutes remarkably reduce the incidence of neurological injury? I do not claim to know the answers to these questions, but they must depend on the shapes of the curves alluded to previously. Ischemic injury, however, is clearly related to multiple variables other than ischemic time such as the preischemic metabolic state (eg, glycogen stores), the methods of preservation, and the physical and pharmacological conditions under which reperfusion occurs. Unfortunately, this manuscript provides no data on these aspects of their technique, nor does it provide any data on the postoperative inotropic requirements of the recipients. Therefore, the excellent results obtained by Backer and co-workers may be due to a multitude of factors with the importance of the technique of operation remaining unknown.

See also page 894.

There may be some technical disadvantages to the technique described by Backer and co-workers. My colleagues and I have not been fortunate enough to acquire donors for the several neonates whom we have listed for transplantation, but my own experience with palliative stage I operations on patients with HLHS leads me to have some concerns about the hemodynamic compromise induced by the manipulations required to place the two right atrial cannulas in the positions described by Backer and co-workers. Also, it seems to me that the risk of having problems with the pulmonary artery anastomosis (particularly the length of the donor pulmonary artery) is greater if this anastomosis is the last one performed. However, Backer and co-workers do not report on any technical problems associated with their technique.

A more global issue is that of the choice of appropriate therapy for patients with HLHS. One unknown in this and other reports of transplantation for HLHS is the number of patients referred to the institution with this diagnosis. It has been our experience that patients with HLHS may be difficult to control medically as pulmonary resistance declines while waiting for an appropriate donor. Therefore, in reports of transplantation for HLHS, one always wonders how many children were either listed for transplantation but died before a donor was available, were declined for transplantation because of inability to be medically managed until a donor became available, or had parents who declined transplantation as an option. It will be necessary in all future reports of transplantation for HLHS to include all of the patients who were seen at that institution (including telephone consultations) with the diagnosis, not just the patients who underwent transplantation.

Despite the favorable results reported with this technique of transplantation, the relative roles of "conventional" stage I palliative operations and transplantation for these patients remains unclear in my own mind. Preliminary (unpublished) data from our own experience suggest that there may be morphological predictors of poor outcome with a stage I approach, and for such a subgroup of patients transplantation may be the best choice. Although the truly long-term (ie, lifetime) outcome of patients after a Fontan operation is unknown, I believe that over the intermediate term, a Fontan circulation is preferable to a lifetime of immunosuppression. As less toxic

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immunosuppressive agents become available, the balance may tip in the direction of transplantation, but until problems with donor availability and immunosuppression toxicity are reduced, we continue to favor the conventional stage I approach for most patients with HLHS. We have employed transplantation for patients who are chronic failures of the conventional approach, and this approach has the advantage of removing some of the urgency to find a donor for the unoperated neonate. However, it fails to address the issues of survival of a stage I operation and the morbidity and mortality between stage I and subsequent "corrective" operations.

Perhaps what is most remarkable of all is that discussions of what is the best surgical option are now occurring for a defect that was considered uniformly fatal only 10 years ago. Backer and co-workers are to be commended for continuing to push forward the limits of our knowledge of the treatment of this defect. One can only hope that health policy planners and third-party payors are not so shortsighted that they will prevent responsible institutions and investigators from continuing to push the limits of therapy as these authors have.

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Surgical Treatment of Wolff-Parkinson-White Syndrome

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Since 1984, Guiraudon and associates from London, Ontario, have advocated an epicardial dissection technique for surgical ablation of accessory pathways responsible for the Wolff-Parkinson-White syndrome. Cardiopulmonary bypass has not been required for accessory pathways located in the right free wall and posterior septal regions of the atrioventricular (AV) groove. The fat pad in the AV groove or posterior septal space is dissected off the *atrium* down to the level of the annulus of the AV valve(s). Cryosurgical lesions are then applied to the annular tissue to ablate any juxtaannular accessory pathways.

Previously, application of this technique to accessory pathways located in the left free wall space was associated with hemodynamic instability owing to dislocation of the heart out of the pericardial space. In virtually all instances, cardiopulmonary bypass was necessary to perform this dissection, which was begun on the *atrial* side of the left AV groove and carried down to the level of the mitral annulus. The circumflex coronary artery and coronary sinus were thus dissected "en bloc" off the left atrial wall; problems with tearing of the friable left atrial tissue or disruption of the AV groove were occasionally encountered, and concerns about proximity of the circumflex coronary artery to the cryoprobe during freezing have been expressed. Finally, even with cardiopulmonary bypass, exposure can be difficult, particularly in patients with enlarged hearts.

See also page 968.

These difficulties have prompted Guiraudon and associates to alter their dissection technique for left free wall pathways. Their paper in this issue of *The Annals* entitled "Surgical epicardial ablation of left ventricular pathway using sling exposure" [1] describes a method of applying an epicardial dissection technique to pathways located in the left free wall space without the need for cardiopulmonary bypass.

To facilitate exposure, the dissection is now performed by dissecting the AV groove fat pad off the *ventricle* instead of the atrium. This permits better access to the AV groove tissue because the mitral annulus can be approached from above rather than from below (as the heart is dislocated from the pericardium). This epicardial dissection from the *ventricular* side of the AV groove was originally reported by Watanabe and associates [2]. A logical extension of this dissection technique has been to devise a method of exposure to dislocate the heart out of the pericardium that seemingly does not cause hemodynamic compromise, thus obviating the need for cardiopulmonary bypass.

It is important to recognize that *both* the dissection technique and the exposure technique have been modified; as before, attempts to perform the older "en bloc" resection technique (dissection off the atrial wall) without cardiopulmonary bypass support are not feasible.

The advantages and disadvantages of the more established endocardial technique versus the "en bloc" or *atrial* epicardial dissection technique have been discussed for the past 5 years. Initially, the advocates of the epicardial technique stated that this approach led to a lower morbidity and mortality than the endocardial approach; in fact, no difference exists between the two techniques when comparable series of patients are analyzed [3, 4]. The one study in which the different techniques were performed in the same institution demonstrated no differences in morbidity or mortality between the techniques [5].

The rationale for developing an alternative approach to the classic endocardial dissection technique for ablation of Wolff-Parkinson-White pathways, as stated by Guiraudon and associates [1], is as follows: avoidance of heparin, which results in bleeding during the dissection and in the postoperative period; avoidance of cardiopulmonary bypass and its associated morbidity; and avoidance of cardioplegic arrest. Each of these is obviously necessary for dissections on the left side of the heart using the endocardial technique.

In the current report [1], unpublished data from their group are referenced to indicate that cardiopulmonary bypass itself is associated with a significant release of the MB fraction of creatine kinase. Although these data suggest that cardiopulmonary bypass is associated with myocardial damage, they are at odds with the generally accepted concepts regarding bypass. Clearly, heparinization of the patient produces increased intraoperative bleeding and increases the risk of postoperative bleeding and complications. However, intraoperative bleeding that interferes with the dissection in the arrested heart is minimal as compared with bleeding in the beating heart; readministration of cardioplegic solution washes out fat debris and red cells that may obscure the dissection field. In the paper by Guiraudon and associates [1], postoperative bleeding is compared between two groups of patients undergoing dissection, one on and one off bypass; no difference in blood loss was present at 24 hours. Our group's experience with the endocardial technique, as it is

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now performed, is that only 2 of 325 patients have required a return to the operating room for postoperative bleeding. Finally, cardioplegic arrest has the advantage of providing a still and bloodless operative field for the dissection, as opposed to dissection on the beating heart; using current techniques of myocardial preservation, the risk of myocardial injury due either to lack of adequate protection or to the cardioplegic solution itself is extremely small.

The requirement of a bloodless, quiescent operative field to allow complete anatomical dissection was one of the changes made by Cox in 1981 from the original endocardial technique described by Sealy, Iwa, and their associates. These changes were made to achieve a singular goal: to increase the success rate of the initial operative procedure to 100%, thereby reducing the reoperative rate to 0%. Alteration of the operative focus from an electrophysiological one to an anatomical one allowed identical anatomical dissections to be performed for each patient once the anatomical region containing the accessory pathway(s) was defined. To achieve the necessary complete anatomical dissection a quiet, dry operative field is optimal. This approach has worked well, even in a patient population in which 20% of patients have *multiple* accessory pathways, an incidence that is significantly higher than other series reported in the literature [4]. The reoperation rate for the epicardial technique remains approximately 3% to 4% in Guiraudon's series [1, 3].

There are two concerns regarding the techniques described by Guiraudon and colleagues in their current paper [1]. The first, which will be answered only by long-term follow-up, relates to the fact that the majority of these patients are young and otherwise healthy with normal coronary arteries. To perform the epicardial "direct" dissection described here, it is necessary to skeletonize the major branches of the circumflex coronary artery as the fat pad is elevated off the top of the ventricular free wall. These marginal branches are epicardial vessels, and if they are not separated from the surrounding AV groove fat pad, a substantial recurrence rate of the arrhythmia can be expected because the accessory pathways frequently accompany the vascular bundles. Because the junction of the epicardial fat pad and the ventricle has to be divided on both sides of the vessel(s) to expose the AV groove and mitral annulus, there may be a long-term risk of fibrosis of the segment of coronary artery that has been stripped of its periadventitial tissue over the length of approximately a centimeter. As with the "en bloc" epicardial technique, concern remains regarding the proximity of the circumflex coronary or its marginal branches to the cryoprobe. It is important to note that with the endocardial approach, which also dissects the AV groove fat pad off the top of the ventricular free wall, the epicardial branches of the circumflex coronary artery remain intact within the epicardial reflection at the completion of the dissection.

The second concern is that the "sling exposure" technique should only be applied with care, if at all, to older patients undergoing operation for ablation of accessory pathways and to patients with clinically significant ventricular dysfunction. Dislocation of the heart for the 40 to 60 minutes necessary to complete the dissection in this higher-risk population would seem unjustified simply to avoid cardiopulmonary bypass, despite undocumented statements in the paper regarding the lack of effect on systolic and diastolic function induced by the sling exposure. Hemodynamic stability is enhanced in all patients undergoing this operative technique by large-volume crystalloid infusion. The 1 patient included in this paper with coronary artery disease had the epicardial technique performed on bypass before revascularization.

The operative morbidity and mortality rates obtained by Guiraudon and associates with this technique are excellent. The failure rate remains higher than that reported for the endocardial dissection technique in similar patients, however, and longer follow-up is needed to assure that occult inadvertent injury of the coronary arteries has not occurred.

How, then, does a surgeon decide which approach to use when he or she is just starting to perform operation for the interruption of accessory pathways? In the final analysis, the answer appears to be based on (1) the intraoperative conditions with which that individual surgeon will be most comfortable and (2) the individual surgeon's assessment of the balance between the extremely low risks of cardiopulmonary bypass and cardioplegic arrest as compared with the low but finite risk of initial-operation failure associated with the epicardial dissection technique. Supraventricular arrhythmia operations have been unique among cardiac procedures in that multiple operations finally resulting in a surgical cure are termed a success.

Regardless of the technique chosen, the results should approach (if not achieve) a 100% initial-operation success rate with an operative mortality in elective uncomplicated cases approaching 0%. These excellent results argue strongly for the consideration of surgical ablation of accessory pathways as the preferred alternative to a lifetime of medical therapy in these predominantly young, otherwise healthy patients.

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Treatment of Postoperative Infection of Ascending Aorta and Transverse Aortic Arch, Including Use of Viable Omentum and Muscle Flaps

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Postoperative infection of the ascending aorta and aortic arch in 40 patients was treated by antibiotic therapy alone (4 patients) or by operation and *lifelong* suppressive antibiotic therapy (36 patients). Complications of infection included antibiotic-resistant infection, infected false aneurysm, rupture of suture line, aortocutaneous fistulas, aortic–right ventricular fistulas, arterial embolus, aortic valve insufficiency, aortobronchial fistula, mediastinal abscess, and chest wall problems. These were treated by a variety of operations including composite valve-graft replacement, graft replacement, patch-graft closure of false aneurysm, simple suture of disrupted suture lines and false aneurysm, and debridement of mediastinum and chest wall. The area of reconstruction was covered, and mediastinal dead space was reduced by mobilization of viable tissue, including local tissue and distant structures such as flaps of muscle and omentum. Thirty-three patients (83%) were early survivors, and 28 patients (70%) were alive and well at last follow-up 4 months to 6.5 years after operation.

(Ann Thorac Surg 1990;50:868–81)

Infection occurring after operation for disease of the ascending aorta and transverse aortic arch is associated with such grave complications as aortic valve insufficiency; peripheral arterial emboli; false aneurysm; rupture into the pericardium, mediastinum, pleural cavity, or heart chamber; chronic draining external sinuses; aortocutaneous fistula; aortobronchial fistula; hemolysis; graft deformity; chronic sepsis; septicemia; shock; and death (Figs 1–7). This segment of aorta is particularly susceptible to the complication of infection because the area of reconstruction lies within a wound that communicates directly with the skin. Thus, wound complications that may be of little importance elsewhere can result in disastrous consequences for patients who have an operation on the ascending aorta or arch.

The reported incidence of infection after abdominal aortic and peripheral arterial operations ranges from 1% to 6% [1–4]. Treatment of the complication at this level has usually comprised insertion of an extraanatomical bypass graft, removal of the infected graft, and temporary antibiotic therapy (1 to 6 months). The reported survival after such operations ranges from 50% to 90% with long-term problems of recurrent infection, arterial occlusion, ischemic symptoms, reoperation, loss of limbs, and death [5–8].

The incidence of infection after ascending aortic and aortic arch operations has not been reported. The principle of graft excision and extraanatomical bypass is not applicable in the treatment of most of these cases. However, the results of treatment by direct methods in the very small number of reported cases [9–13] have encouraged and guided our approach to this entity. The purpose of this report is to present the senior author's (E.S.C.) overall experience with postoperative infection at this level and its treatment by in situ operation and *lifelong* suppressive antibiotic therapy as previously reported for treatment of mycotic aortic aneurysm [14].

Material and Methods

During the 7-year period between June 1, 1982, and August 21, 1989, 652 patients without infection were treated by graft replacement of the ascending aorta, the aortic arch, or the ascending aorta and transverse aortic arch. In 21 (3%) of these patients, infection developed in or about the area of operation and became clinically suspected at periods ranging from 1 week to 2 years. In addition, 19 patients were referred from other hospitals for well-established chronic infection of the ascending aorta or arch. Therefore, the total population in this retrospective study is 40 patients, and they are considered as a single unit. There were 26 men and 14 women ranging in age from 21 to 75 years (average age, 55 years).

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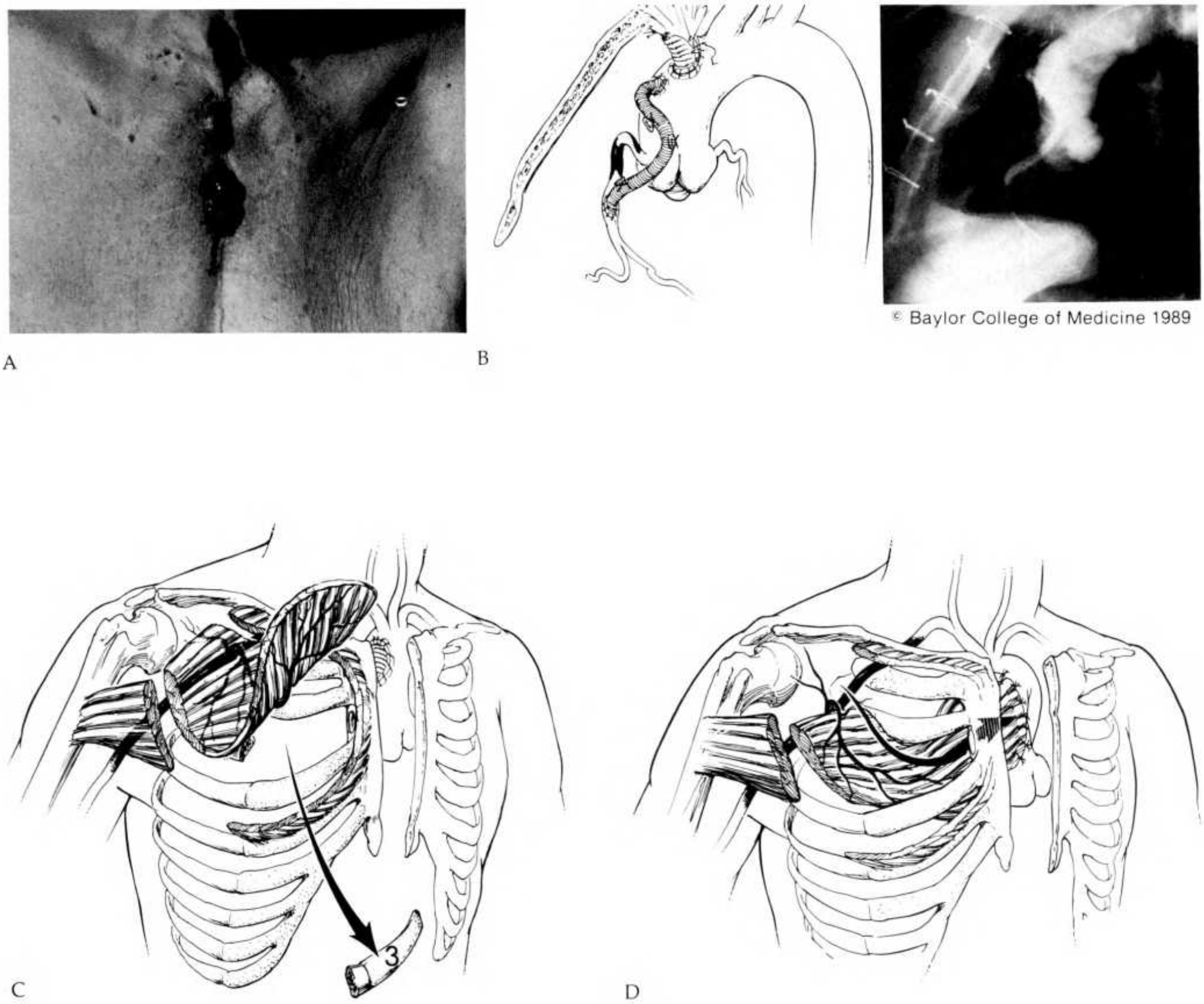

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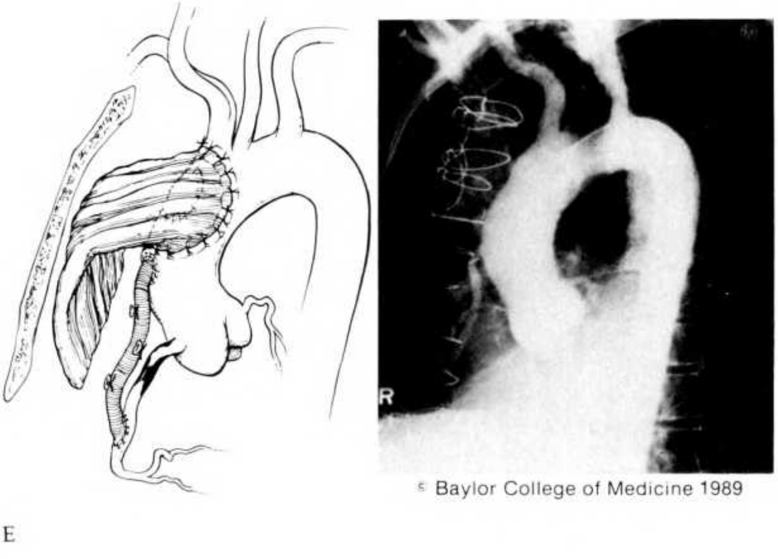

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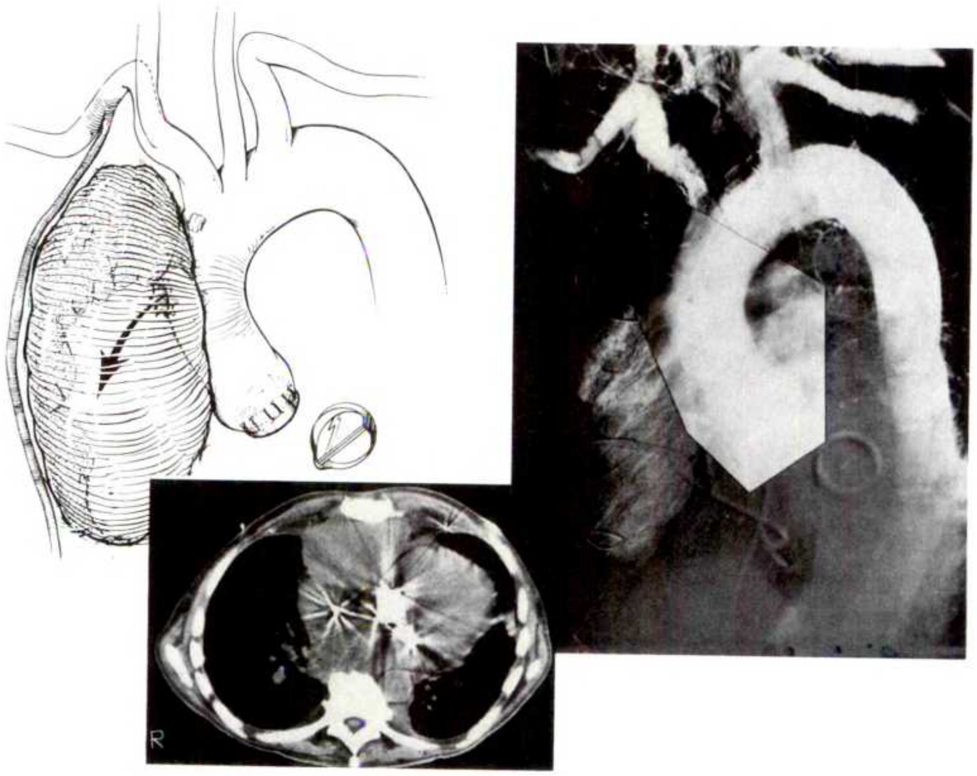
Fig 1. Patient with aortocutaneous fistula occurring after coronary artery bypass grafting. (A) Anterior chest wall has a healing external aortocutaneous fistula opening in the upper end of the old scar and a fresh external opening with protruding granulation tissue just below it occurring for the fifth time 3½ years after coronary artery bypass grafting and one previous attempt at correction. (B) Drawing and aortogram show a false aneurysm arising from the site of previous patch-graft repair of infected false aneurysm, which arose from the aortic cannulation site, which extends to the anterior chest wall opposite the latter skin defect. (C) A large segment of the right pectoralis major muscle was completely mobilized, preserving its nerve and blood supply. (D) The mobilized muscle was transferred into the chest through a window created by removing the medial end of the third rib and costal cartilage. (E) The mobilized muscle was sutured around the ascending aorta, which was repaired by debridement and reinsertion of a new Dacron patch (drawing), thus producing a satisfactory aortic reconstruction (aortogram). The patient continues to do well 4.5 years after operation.



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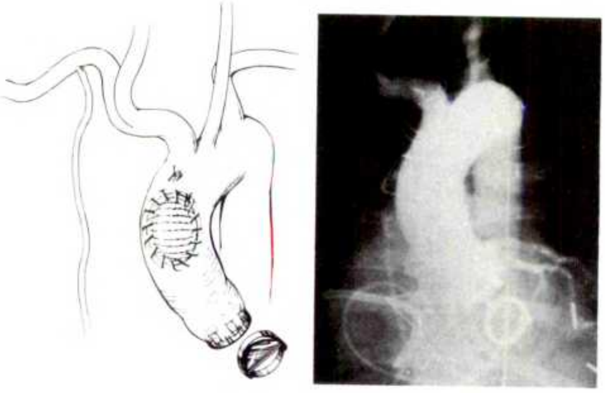

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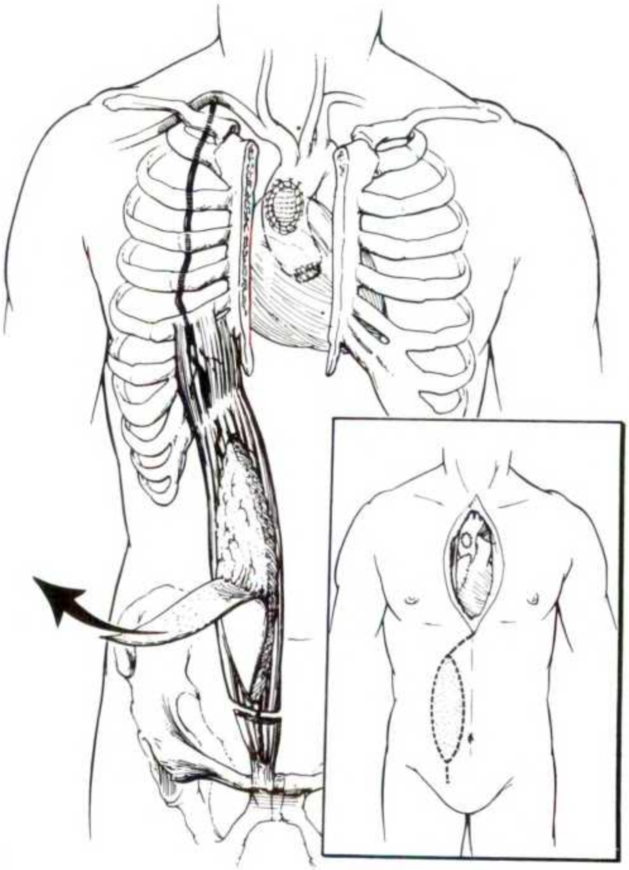

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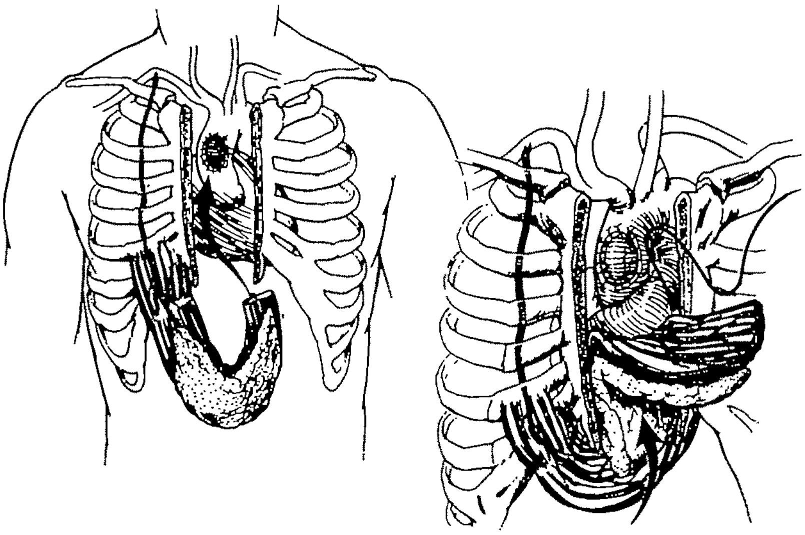
Fig 2. Patient with large retrosternal false aneurysm at admission 7 months after replacement of the aortic and mitral valves and 11 operations for bleeding or infection. (A) Drawing, computed tomographic scan, and aortogram show location, nature, and extent of disease. (B) With circulation arrested, the chest was entered, and the aneurysm site was debrided and closed with a large Dacron patch. (C) A musculocutaneous flap was created to cover the aorta and fill the mediastinal dead space. (D) The flap was introduced into the mediastinum, thus covering the reconstructed aorta. (E) The bulky segment of flap filled the dead space, and the wound was closed. The patient is working and doing well 18 months later.



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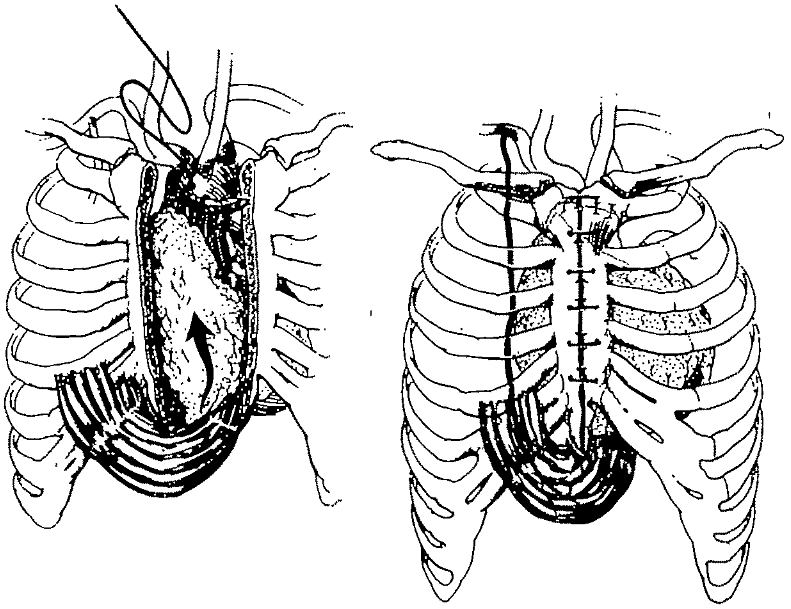

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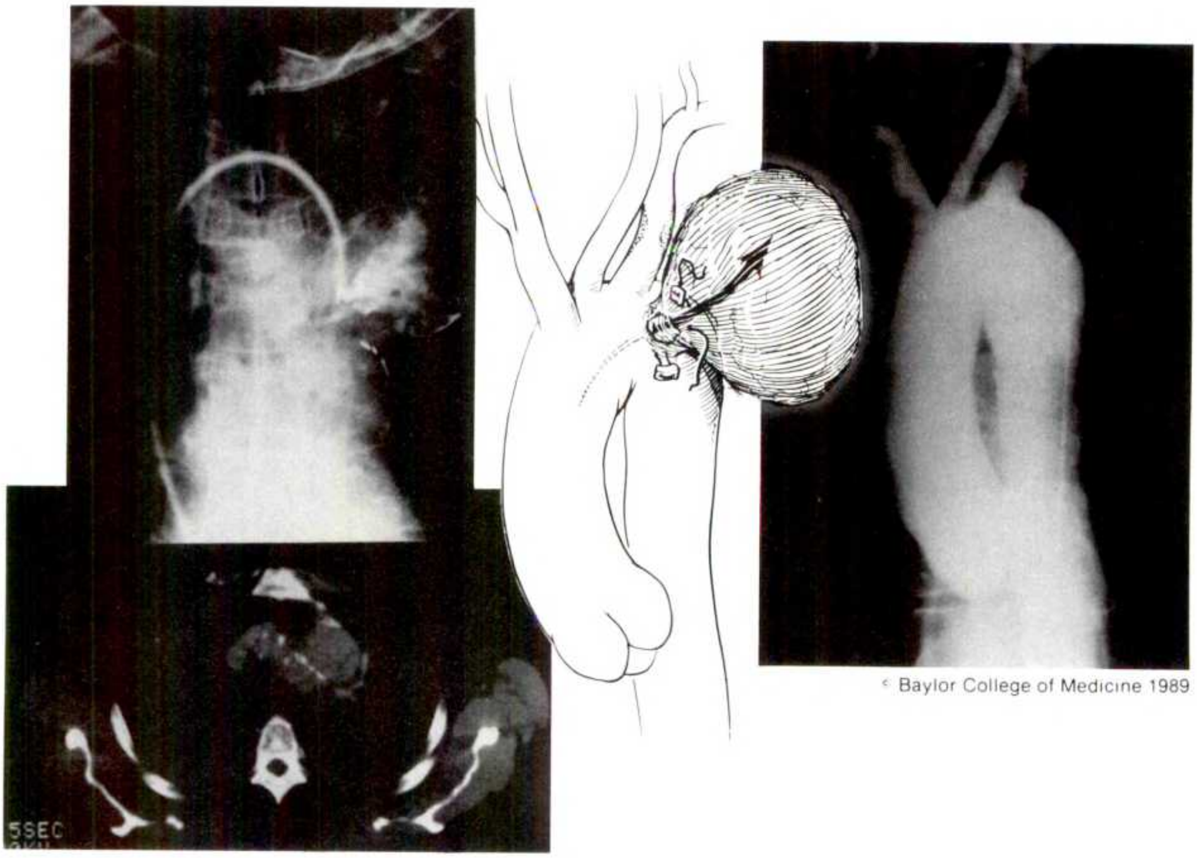
Fig 2. Continued.

Original Operation

The original operations preceding aortic infection are shown in Table 1. In patients whose operations were performed elsewhere and did not require grafts, such as valve replacement and coronary artery bypass grafting, infection occurred at sites of aortic repair, aortic cannulation, vein graft attachment, and aortic cardioplegia perfusion sites (see Figs 1, 2). Dacron patch grafts or Teflon pledgeted sutures had been employed either at the time of the original operation or at the time of subsequent repair. In 1 patient, infection of the lesser curvature of the distal aortic arch resulted from aortic injury during an attempt at lymph node biopsy from a small anterior intercostal incision (Chamberlain procedure) (see Fig 3) and in another, at the site of graft replacement for coarctation. Both of these patients required repair that extended proximally into the transverse aortic arch and, thus, are included in this group of patients with infection involving the ascending aorta and transverse aortic arch.

Predisposing Factors

A variety of factors contributed to the development of graft, perigraft, or native aortic infection. Some patients had multiple factors. The most common factor was wound-related problems. Suppuration in the subcutaneous tissues, sternal wound dehiscence, and drainage of infected mediastinal fluid were present in 13 patients (see Figs 4, 5). Thirteen patients had undergone two to 11 previous operations for cardiac, aortic, or other disease, and some of these patients had had more than one operation during the same hospitalization, the later procedures performed through recently made incisions for complication of the previous operation. Median sternotomy wounds were not closed for several days after the



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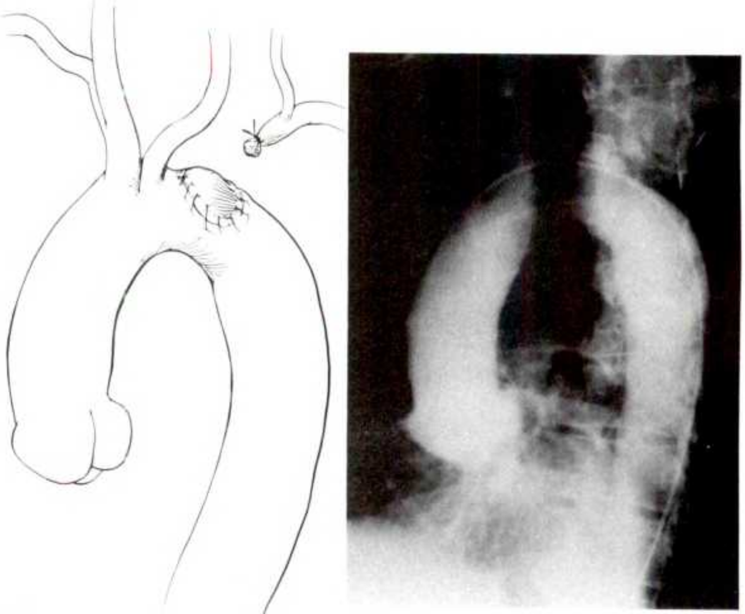

Fig 3. Patient with aortocutaneous fistula and false aneurysm in lesser curvature of the distal aortic arch 13 months after aortic injury during a Chamberlain procedure. (A) Percutaneous angiogram (upper left) performed through the chronic aortocutaneous sinus tract, computed tomographic scan (bottom left), and drawing show the false aneurysm and route of the aortocutaneous fistula. Aortogram (right) made before operation was nondiagnostic. (B) Drawing and aortogram made after operation show satisfactory aortic reconstruction using the left subclavian artery as described by Waldhausen and Nahrwold [18] for repair of coarctation. The patient did well until his death from myocardial infarction 6.5 years later.

B

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original operations in 4 patients. Distant infection in the lungs, in the urinary tract, and in wounds other than a median sternotomy was present in 7 patients. Line sepsis (3 patients), tracheotomy (2 patients), or systemic lupus erythematosus (1 patient) requiring long-term steroid therapy was present in 6 patients. Aortobronchial fistula was present before the initial operation in 1 patient.

Microbiology

Seven (18%) of the patients were treated throughout their illness with antibiotics before culture studies. Neither bacteria nor fungi were grown in cultures from blood, wound, or urine specimens or from material removed at operation in these patients. Clinical signs of infection

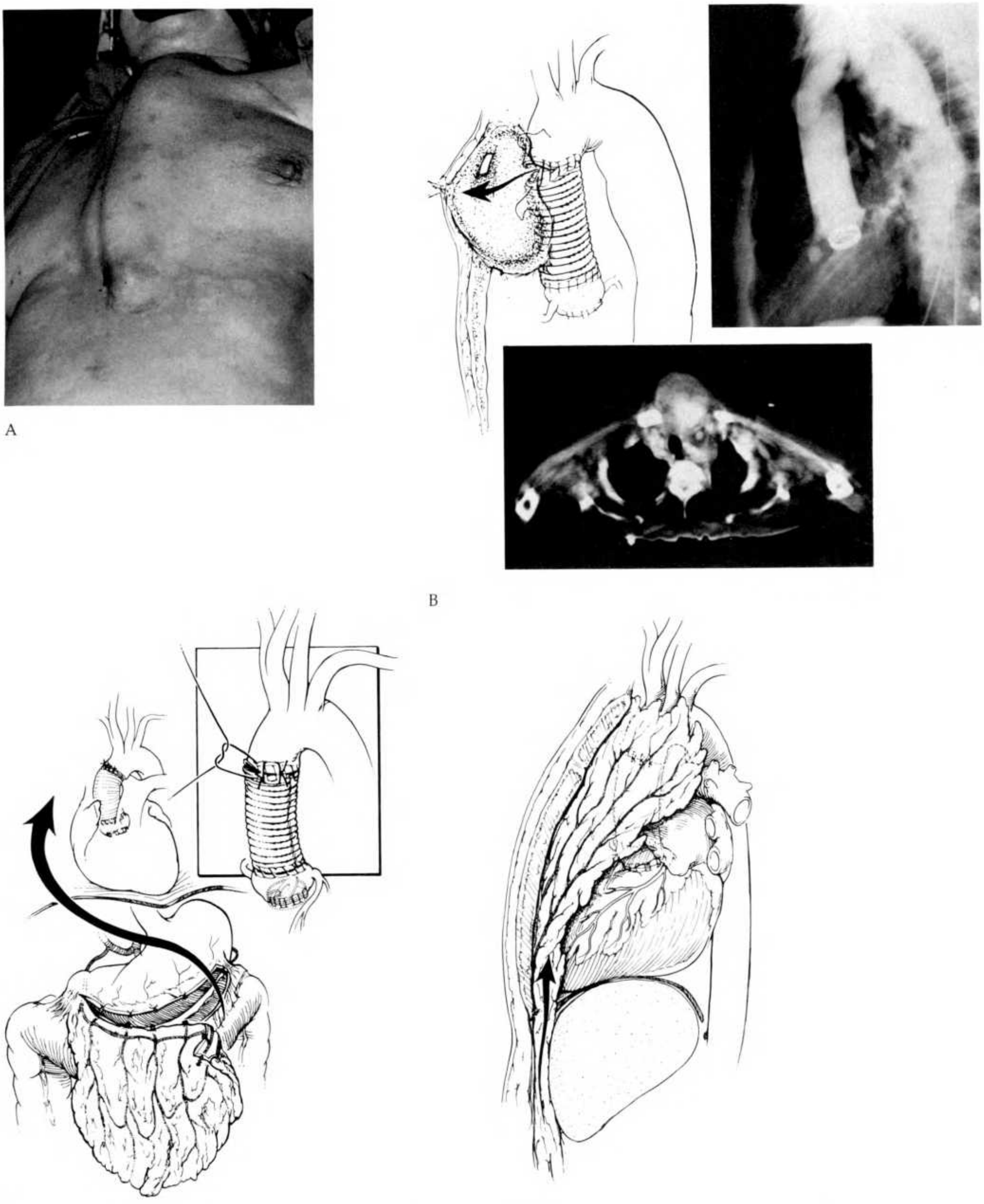

Fig 4. Patient with a subcutaneous pulsatile anterior chest wall mass 9 weeks after composite valve-graft replacement of the ascending aorta and 4 weeks after sternal debridement and chest wall closure with pectoral muscle flaps. (A) The defect in the lower end of the scar is the site of multiple episodes of projectile bleeding (aortocutaneous fistula). (B) Drawing, computed tomographic scan, and aortogram show large false aneurysm protruding through the chest wall and displacing the ascending aorta inward. (C) Method of treatment included debridement and repair of disrupted distal suture line by simple suture, and mobilization of the greater omentum from the stomach and colon, with circulation maintained through the right gastroepiploic artery and vein. (D) Transfer of omentum into chest to fill dead space and to cover graft and repaired ascending aorta. The patient did well until her death 2 years later from myocardial infarction

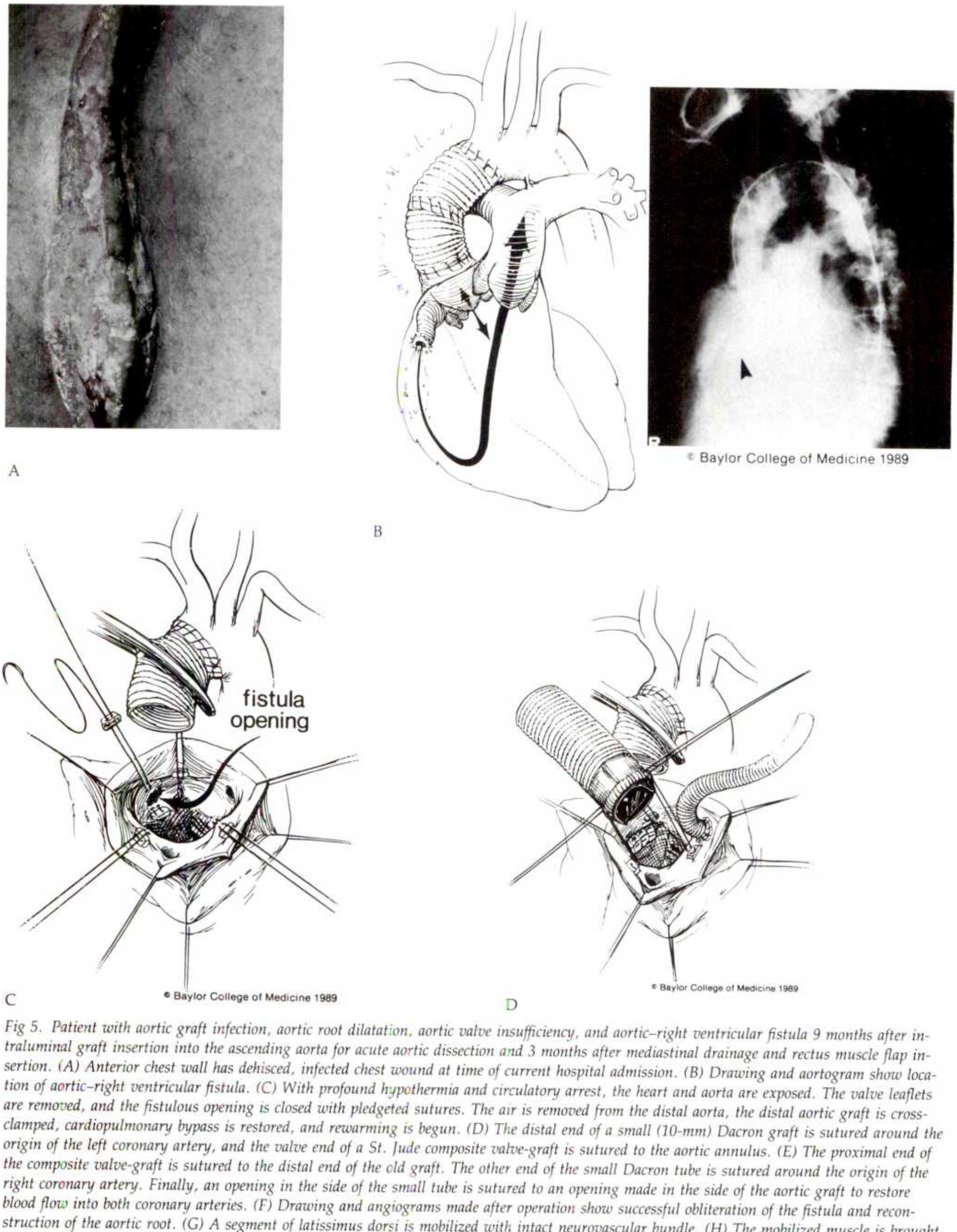

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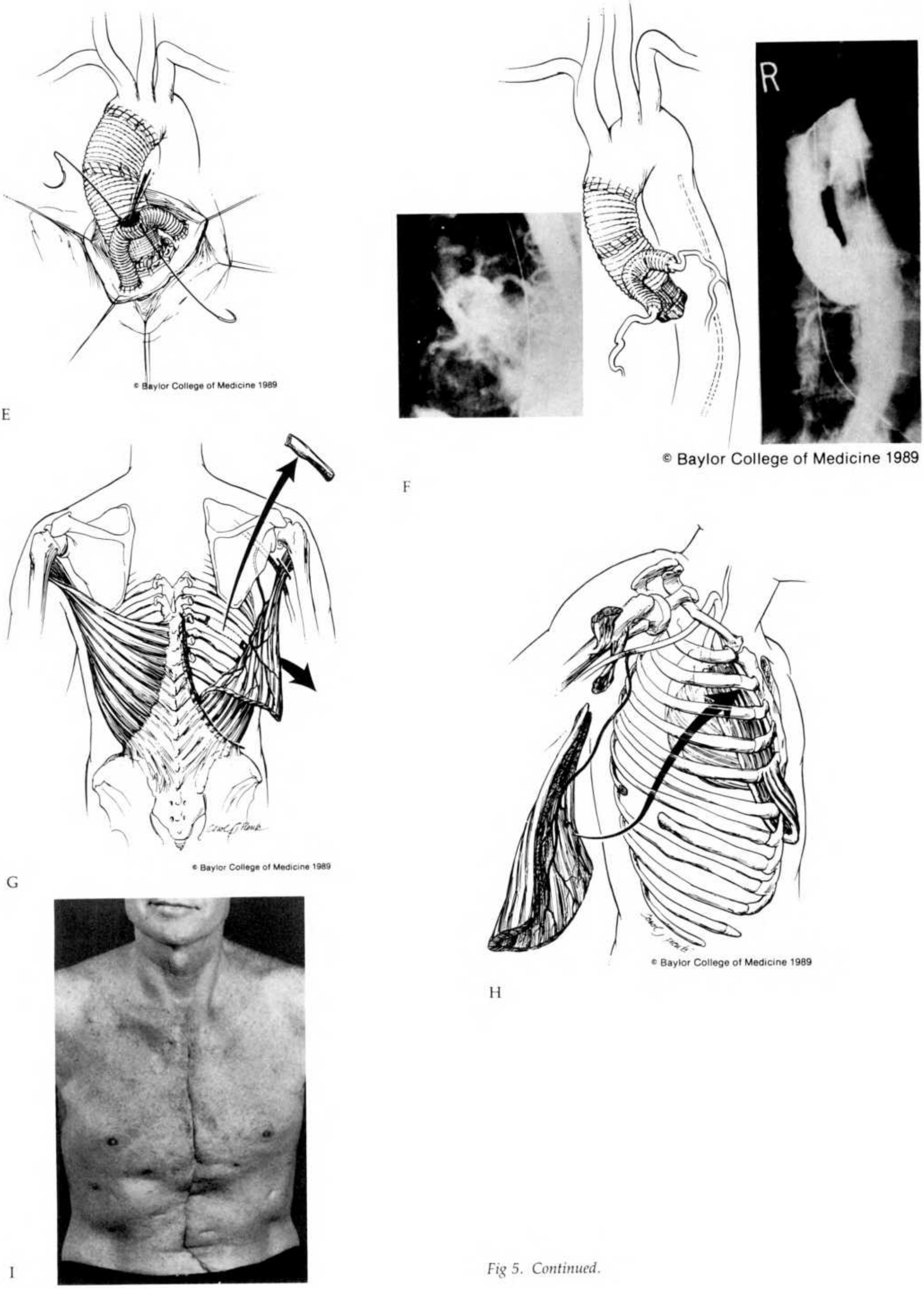
Fig 5. Patient with aortic graft infection, aortic root dilatation, aortic valve insufficiency, and aortic–right ventricular fistula 9 months after intraluminal graft insertion into the ascending aorta for acute aortic dissection and 3 months after mediastinal drainage and rectus muscle flap insertion. (A) Anterior chest wall has dehisced, infected chest wound at time of current hospital admission. (B) Drawing and aortogram show location of aortic–right ventricular fistula. (C) With profound hypothermia and circulatory arrest, the heart and aorta are exposed. The valve leaflets are removed, and the fistulous opening is closed with pledgeted sutures. The air is removed from the distal aorta, the distal aortic graft is cross-clamped, cardiopulmonary bypass is restored, and rewarming is begun. (D) The distal end of a small (10-mm) Dacron graft is sutured around the origin of the left coronary artery, and the valve end of a St. Jude composite valve-graft is sutured to the aortic annulus. (E) The proximal end of the composite valve-graft is sutured to the distal end of the old graft. The other end of the small Dacron tube is sutured around the origin of the right coronary artery. Finally, an opening in the side of the small tube is sutured to an opening made in the side of the aortic graft to restore blood flow into both coronary arteries. (F) Drawing and angiograms made after operation show successful obliteration of the fistula and reconstruction of the aortic root. (G) A segment of latissimus dorsi is mobilized with intact neurovascular bundle. (H) The mobilized muscle is brought through an opening in the chest wall and sutured around the Dacron graft. Operation is completed by wound closure. (I) Photograph made 6 months after operation shows well-healed wound. The patient continues to do well 5 years after operation.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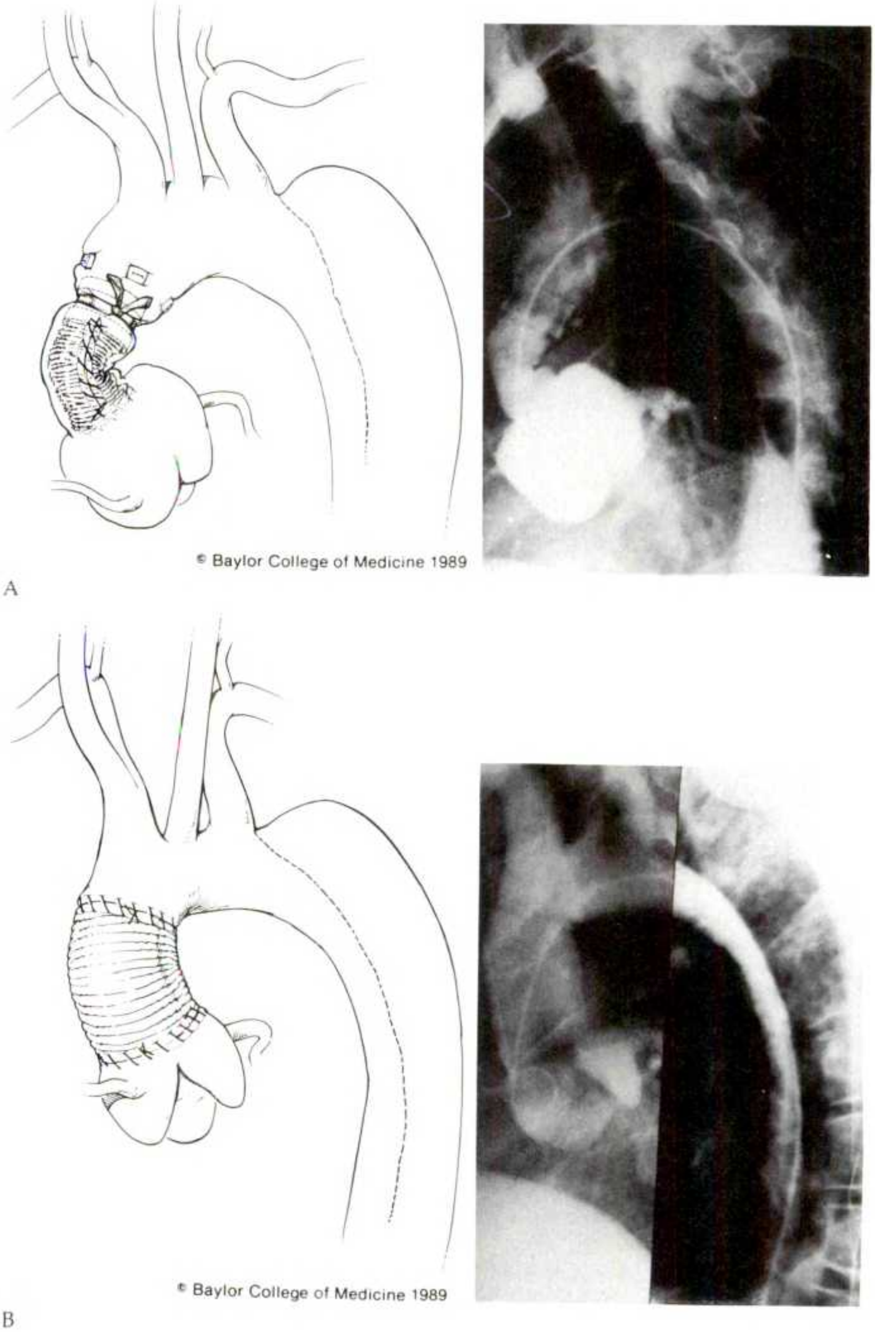

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Fig 6. Patient with infected, deformed intraluminal graft inserted 8 months earlier for acute dissection of aorta. (A) Drawing and aortogram show kinked, stenotic, and partially obstructed intraluminal graft and aortic valve insufficiency. Blood studies revealed elevated levels of serum bilirubin and lactate dehydrogenase, elevated reticular cell count, and anemia, indicating hemolysis. (B) Drawing and aortogram made after operation show unobstructed graft and competent resuspended aortic valve apparatus. The graft site was covered with mobilized thymic and pericardial fat. The patient continues to do well 2 years after operation.

were clearly evident during the course of the disease and included fever, chills, false aneurysm, and other anatomical evidence of the presence of infected operative sites.

Serial blood and wound cultures and cultures of tissue or graft material removed at operation were positive in the remaining 33 patients. As shown in Table 2, cultures grew gram-positive bacteria in 23 patients (58%), gram-negative bacteria in 9 patients (23%), and *Candida* sp in 1 patient who also had gram-positive organisms. *Staphylococcus* species was the most common organism in the first group and *Pseudomonas aeruginosa* in the second group. Finding *Salmonella* sp in 1 patient was a surprise and suggested the possibility that the disease was preexistent or intercurrent in this patient despite the absence of other symptoms.

The bacteria were sensitive to intravenous antibiotics in all patients and to oral antibiotics in all but 1 patient with *P aeruginosa*. The bacteria in that patient were moderately sensitive to oral trimethoprim and sulfamethoxazole, and this was prescribed until a more effective antibiotic (quinolone) was marketed. The *Candida* infection responded well to amphotericin B given intravenously and has been suppressed for 18 months with oral ketoconazole.

Complications of Infection

Multiple complications were frequently present in the patients requiring operation. Twenty-eight patients had 41 cardiac or aortic complications. These included infected false aneurysm in 19 (see Figs 1–3); rupture of infected

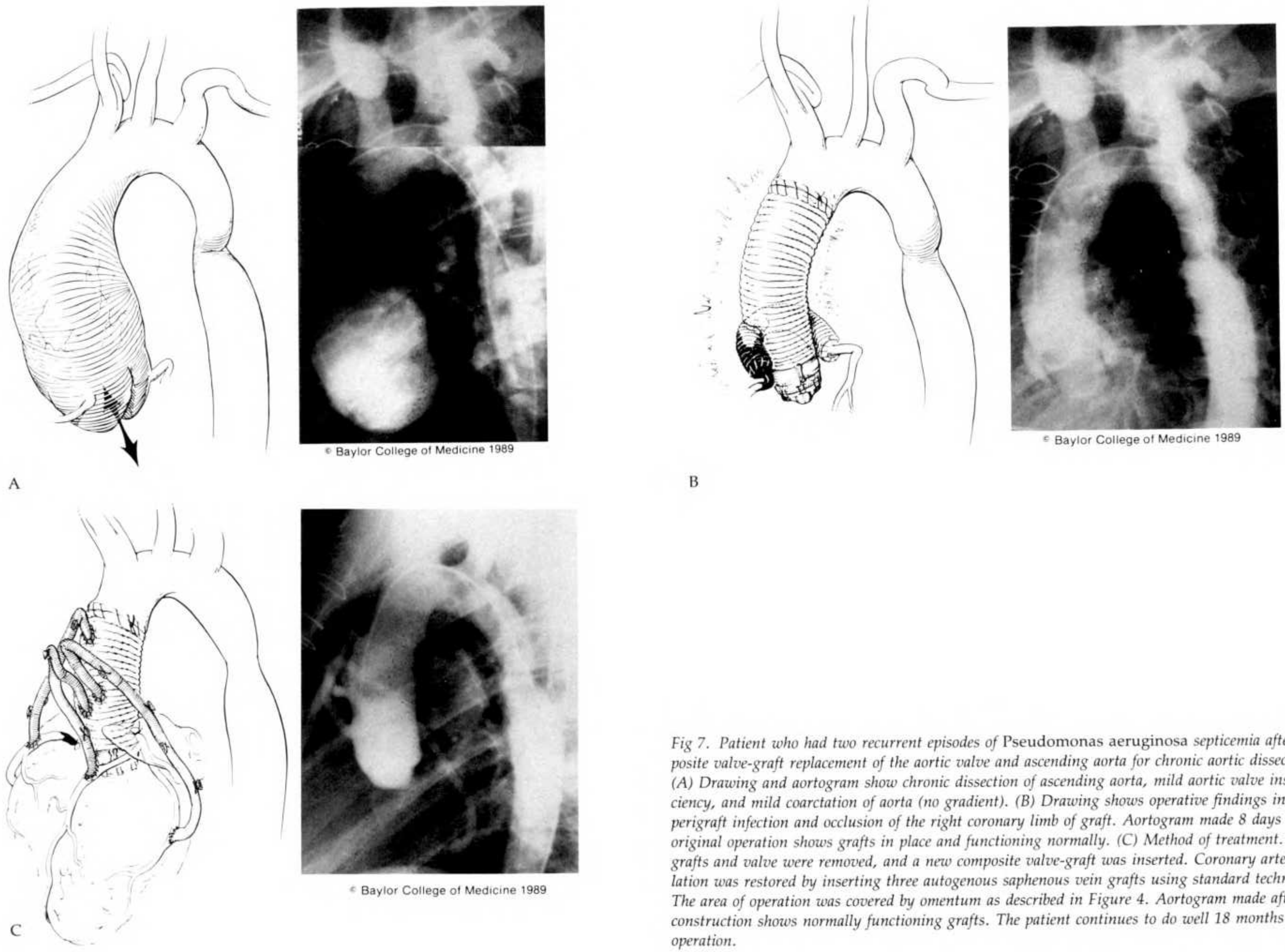

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Fig 7. Patient who had two recurrent episodes of Pseudomonas aeruginosa *septicemia after composite valve-graft replacement of the aortic valve and ascending aorta for chronic aortic dissection. (A) Drawing and aortogram show chronic dissection of ascending aorta, mild aortic valve insufficiency, and mild coarctation of aorta (no gradient). (B) Drawing shows operative findings including perigraft infection and occlusion of the right coronary limb of graft. Aortogram made 8 days after original operation shows grafts in place and functioning normally. (C) Method of treatment. The old grafts and valve were removed, and a new composite valve-graft was inserted. Coronary artery circulation was restored by inserting three autogenous saphenous vein grafts using standard techniques. The area of operation was covered by omentum as described in Figure 4. Aortogram made after reconstruction shows normally functioning grafts. The patient continues to do well 18 months after operation.*

Table 1. Operations Preceding Infection Complication

Procedure	No. of Patients[a]
AVR, ascending aortic replacement[b]	14 (35)
AVR, ascending aortic and aortic arch replacement[b]	7 (17.5)
Ascending aortic replacement	7 (17.5)
Ascending aortic and aortic arch replacement	3 (7.5)
Aortic arch replacement	1 (2.5)
Distal arch repair with graft	1 (2.5)
Coarctation repair with graft	1 (2.5)
Biopsy lesser curve, aortic arch with Dacron patch closure	1 (2.5)
Redo AVR	2 (5)
AVR	1 (2.5)
AVR, MVR	1 (2.5)
Coronary artery bypass grafting	1 (2.5)
Total	40 (100)

[a] Numbers in parentheses are percentages. [b] Procedures were done with and without coronary artery bypass grafting.

AVR = aortic valve replacement; MVR = mitral valve replacement.

suture lines in 3 (see Fig 4); aortocutaneous fistulas in 5 (see Figs 1, 3, 4); aortobronchial fistula in 1; aortic–right ventricular fistulas in 2 (see Fig 5); arterial embolus in 2; aortic graft deformity, aortic obstruction, and hemolysis in 2 (see Fig 6); and aortic valve insufficiency in 7 (see Fig 5, 6). In addition, chest wall problems including complete wound dehiscence (see Fig 5), draining sinuses (see Figs 1, 3, 4), osteomyelitis of the sternum, and chondritis of the costal cartilages were present in 13 patients (see Figs 2, 4, 5) and induration and periaortic abscess formation, in 9 patients (see Figs 6, 7). Microscopic or gross evidence of graft infection was found in all of the patients in whom prosthetic grafts were employed originally (see Figs 6, 7).

Table 2. Bacteriology

Organism	No. of Patients[a]
Gram-positive organisms	23 (57.5)
Staphylococcus epidermidis	12
Staphylococcus aureus	8
Enterococcus	1
Corynebacterium species	1
Gram-negative organisms	9 (22.5)
Pseudomonas aeruginosa	7
Proteus species	1
Salmonella species	1
Fungus	
Candida species[b]	1 (2.5)
No growth	7 (17.5)
Total	40 (100)

[a] Numbers in parentheses are percentages. [b] This patient also had cultures that grew *Staphylococcus aureus*.

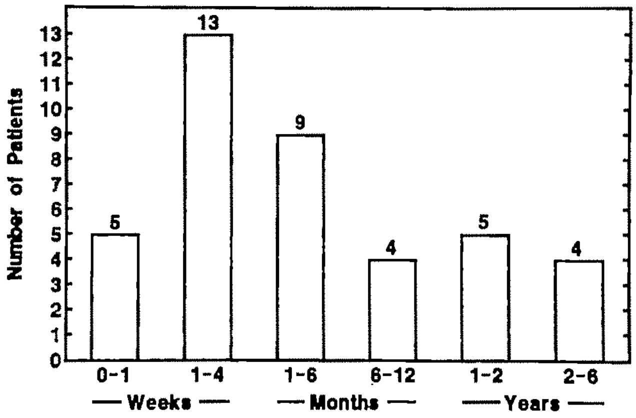

Fig 8. Interval between initial operation and clinical onset of infection.

The interval between the original operation and the appearance of the infectious complication ranged from 1 week to 5.5 years (Fig 8). The number of prior operations performed in the treatment of infectious complications in the 19 patients referred to us with this problem ranged from two to 11. The long interval between original operation and infection in some patients and the complication of peripheral emboli in others suggested bacteremia and bacterial endocarditis as an intercurrent infection unrelated to the original operation. This assumption may be entirely correct; however, the offending organism in most of the patients with long latency was *Staphylococcus epidermidis*, and this organism is well known for this characteristic [15].

Methods of Study

Attempts were made to establish the presence and type of infection by culture of blood, urine, and wound drainage specimens and of surgical samples, including tissue and foreign substances, removed at operation. Antibiotic-sensitivity studies were performed for each bacterium identified. Bacterial specimens were stored for possible future use in determining sensitivity to new antibiotics as they became available or as they were needed. Broad-spectrum antibiotics that included activity against methicillin sodium–resistant *S epidermidis* were given to patients with negative cultures.

False aneurysms and collections of blood clots were determined by computed tomographic scan, magnetic resonance imaging, aortography, and digital subtraction angiography (see Figs 1–7). Perigraft collections of exudates were determined by computed tomographic scanning or magnetic resonance imaging, and aortic valve function was studied by echocardiography. Origins of false aneurysms and their relationship to coronary artery origins or coronary artery graft origins were determined by left heart catheterization and cineangiography.

Treatment

Treatment varied with the acuteness of infection, the bacterial etiology, the complications produced by the

Table 3. Treatment

Method of Treatment	No. of Patients	Deaths 30-Day	Deaths Late
Composite valve-graft replacement	9	3	2
Graft replacement	7	0	0
Patch-graft closure of false aneurysm	4	0	1
Suture of false aneurysm	8	1	4
Perigraft and mediastinal debridement	8	1	1
Antibiotics (medical only)	4	0	1
Total	40	5	9

infectious process, and the response to medical therapy. In general, medical treatment consisted of large doses of appropriate parenteral antibiotics determined by culture and sensitivity studies and given intravenously for 4 (*Streptococcus* and gram-negative organisms) to 6 (*Staphylococcus*) weeks, and then suppressive oral antibiotics were taken daily *for the rest of the patient's life*, as prosthetic heart valves or prosthetic graft materials were present in all patients at the time of our treatment of infection or were employed during our operation for treatment of infection.

Medical treatment alone as just described was attempted in 7 (33%) of our 21 patients and was successful in 4 (19%). Operation was thus performed in 36 patients, 17 of our own and 19 referred to us because of chronic infection. Surgical treatment was combined with the same antibiotic program as used in medical treatment, but the timing of operation was determined by the urgency of the complication. Most operations were performed urgently, ie, within 1 day to 3 days of admission or development of the complication. Intravenous antibiotic treatments were started before operation, changed if indicated by operative cultures, and completed in the period after operation.

For all operations, the principle of "in place" maintenance of circulation was employed. The in situ procedures used in the treatment of these patients are shown in Table 3. Cardiopulmonary bypass using a pump-oxygenator was required in 30 (83%) of the 36 patients. Perfusion was by femoral vein to femoral artery, and profound hypothermia and circulatory arrest was required in 19 patients (53%) [13, 16, 17]. Arrest of circulation was achieved in 12 patients before the chest was entered to prevent fatal exsanguination [13, 16, 17].

Operation consisted of perigraft, mediastinal, and chest wall debridement, graft coverage with viable tissue, and chest closure in 8 patients. Large false aneurysms at suture lines between graft and aorta or graft and aortic annulus were treated by clot evacuation and simple suture of the dehisced suture line in 8 patients in whom the aortic graft and prosthetic aortic valve were functioning normally (see Fig 4). Graft replacement was performed in 7 patients who had deformed or abnormally functioning grafts (see Fig 6). Large false aneurysms of the ascending aorta or arch occurring at sites of perfusion, cardioplegia administration, aortotomy closure, and vein graft anastomosis were treated by evacuation of hematoma, removal of previous foreign material used either in the initial repair or at a subsequent procedure, debridement of the aorta, and reclosure of the aorta using a larger Dacron patch in 3 patients and the left subclavian artery in 1 patient (see Figs 1–3) [18].

Composite valve-grafts were removed and replaced in 2 patients, 1 with peripheral arterial embolus and both with recalcitrant infection. The coronary arteries were reattached in these patients using aorta–distal coronary artery saphenous vein grafts (see Fig 7). Composite valve- grafts were used to replace infected ascending aortic grafts in 7 patients [19]; 5 had separate aortic valve replacement with peripheral embolic complications in 1 and aortic–right ventricular fistula in 1, and 2 had residual aortic root aneurysms, 1 of whom had aortic–right ventricular fistula (see Fig 5).

Additional procedures employed in these patients included coverage of the involved aorta, closure of dead space, and mediastinal antibiotic irrigation. Aortic coverage and closure of dead space was accomplished by mobilizing grossly uninfected local tissue including thymus, pericardial fat pads, muscle (pectoralis major, rectus abdominis, and latissimus dorsi), a combination musculocutaneous flap (rectus abdominis muscle with overlying fascia, subcutaneous tissue, and denuded epithelium), and greater omentum. Local tissues were used in 18, omentum in 9 (see Fig 4), muscle only in 8 (see Figs 1, 5), and musculocutaneous flaps in 2 patients (see Fig 2). Omental flaps based on the right gastroepiploic artery and vein were preferred when omentum was available in adequate size as in patients in a nondebilitated condition and in patients without a previous abdominal operation.

Results

Thirty-five patients (88%) survived 30 days, 4 treated medically and 31 (86%) of the 36 requiring operation. The causes of the five early deaths were coagulopathy and hemorrhage at the time of operation in 2 patients and cardiorespiratory and renal complications in 3. Two additional surgically treated patients died at 2 months while still in the hospital, 1 of renal failure and the other of pulmonary insufficiency, complications arising from the original operation. Thus, 33 patients (83%) including 29 (81%) treated surgically were early survivors and were able to resume normal lives.

Follow-up, ranging from 3 months to 6.5 years, was complete for all patients, and 28 (70%) were alive and well at 4 months to 6.5 years (Fig 9). Two patients treated medically discontinued the oral antibiotics at 1 year and had recurrence of fever and chest pain. Both have remained well since resuming antibiotics. One medically treated patient died 2 years after operation of an atherosclerotic stroke. The cause of the six late deaths in the surgically treated patients was cardiac related in 5 patients and pulmonary disease in 1 patient. Infection has not recurred in any of the surgically treated patients, and all have been faithful to their medical treatment.

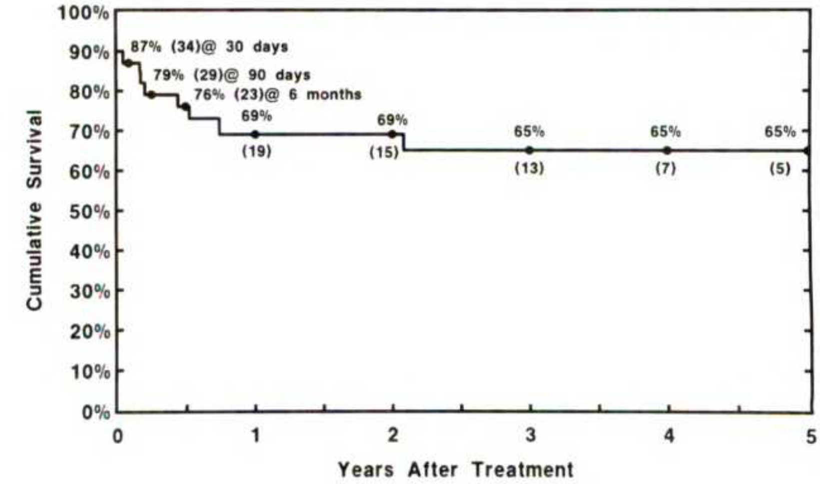

Fig 9. Kaplan-Meier survival probability after operation for infection. Survival from time of treatment is shown (n = 40).

Comment

Postoperative infection of the operative site in patients who have ascending aortic and aortic arch operations is more common than at other aortic locations in the thorax. This difference is explained in part by the fact that the aortic operation lies in a wound that communicates with the skin. Thus, chest wound complications associated with infection in this location, like femoral wound complications in patients with aortofemoral bypass operations, are more likely to lead to postoperative aortic infection. This emphasizes the importance of such prophylactic measures as demanding uninfected skin of the anterior chest wall before operation and meticulous wound care at the time of operation and during the postoperative period. Such care includes minimal use of wound cautery, avoidance of bone wax, complete hemostasis, careful sternal fixation, and wound closure without leaving dead space or strangulated tissue. The wound should be cleaned daily and remain covered until healed or certainly as long as the patient remains intubated either with an endotracheal tube or tracheostomy.

Appropriate antibiotic prophylaxis also plays an important role in minimizing tube graft infection by skin staphylococci, both *Staphylococcus aureus* and *S epidermidis*. Prophylactic antibiotic therapy must be initiated before operation to ensure that adequate blood and tissue levels of the appropriate bactericidal antibiotic will be present at the time of graft or graft and valve insertion. This therapy is continued for 2 to 3 days or until all tubes have been removed. The precise prophylactic antibiotic to be used is determined by the nature of the organisms commonly cultured in the hospital in which the operation is performed. In some hospitals, the penicillinase-resistant, semisynthetic penicillins (methicillin, oxacillin, or nafcillin sodium) are appropriate. Cefamandole nafate or cefuroxime sodium are preferable in hospitals that have a high incidence of methicillin-resistant *S epidermidis*.

We have observed that it is not always necessary to remove grafts in other locations when they become infected provided they are patent and functioning satisfactorily without evidence of false aneurysm or erosion. The infection can be controlled indefinitely in such instances when it is caused by an organism sensitive to a bactericidal antibiotic that can be given in large doses intravenously at first and then continued indefinitely as an oral preparation once the infection is brought under control. We consider the latter the most important principle in the concepts employed in the treatment of the patients in this report. Graft removal was necessary only when the graft was abnormal, when the infection was resistant to antibiotics, when the graft inadequately replaced the original disease, or when it was associated with aortic valve insufficiency or aortocardiac fistula. Moreover, the principle was extended to patients with ruptured suture lines and simple false aneurysms by debridement and closure by simple suture or insertion of patch grafts rather than by graft replacement. We also recognize the importance of graft coverage and elimination of dead space using distant tissue flaps when necessary.

In summary, we believe that the results obtained in these patients justify the use of in situ operations, and their value is evidenced by the fact that local excision and extraanatomical bypass grafting is impossible as an alternative in most instances. Finally, *lifelong* suppressive antibiotic therapy must be combined with operation in every instance.

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DISCUSSION

DR RICHARD E. CLARK (President): It seems these are incredibly difficult cases and this is a superb series with a very small early mortality rate. How did you define early versus late mortality?

DR COSELLI: We were indeed fortunate in this complex and critically ill group of patients. We defined early mortality as 30 days. During this early period 5 patients died, 4 from the medical group and 1 of those treated surgically. Two additional surgically treated patients died while still in the hospital in greater than 2 months, 1 of renal failure and the other of pulmonary insufficiency. Twenty-nine patients (80%) treated surgically survived hospitalization and resumed normal lives.

DR HOOSHANG BOLOOKI (Miami, FL): I agree with you that each one of these is a single case on its own, and one cannot set a rule that can be followed for these patients as a group. Our experience is similar to yours in that all infected ascending thoracic aortic aneurysms are managed with circulatory arrest and hypothermia and with graft replacement followed with antibiotic therapy for life. I got lost in your presentation, however, as to whether you are presenting only infected aneurysms or cases of false (noninfected) aneurysms as well. In other words, did all these patients have positive blood cultures or did some simply have infection at the skin level near the fistula? Also, please tell us how you managed the infected aortic tissue; how much debridement was done?

DR COSELLI: The patients presented represent a combination of both infected false aneurysms and infection involving recurring aneurysms. Clinical signs of infection were clearly evident in all patients during the course of their disease. In addition, anatomical evidence of infection at the operative site was present in all patients who underwent exploration and operation. Seven patients (17%) had negative cultures due to prior antibiotic administration. The remaining 33 patients had positive cultures from blood, wound, tissue, or graft material removed from the operative site. Although placement of graft material in situ in such cases is feasible, lifelong suppressive antibiotic therapy is an inseparable part of their overall management.

Facile Retrograde Cardioplegia: Transatrial Cannulation of the Coronary Sinus

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Joseph S. McLaughlin, MD, and Leonard L. Bailey, MD

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The benefits of coronary sinus (CS) cardioplegia are well known, yet CS cardioplegia is not used widely owing to the need for bicaval cannulation, snares, and an atriotomy. We designed and used in 225 consecutive patients a catheter containing a flexible removable stylet that, when shaped into a hockey-stick configuration, enabled blind intubation of the CS through a small pursestring in the right atrium, guided easily into the CS using a finger on the atrioventricular groove at the inferior vena cava. The CS was intubated in all patients; a pressure-limited balloon at the catheter tip was inflated after cross-clamping. An integral distal pressure line measured CS pressure. Catheters were placed distally within the great cardiac vein beyond the posterior interventricular vein; the position did not alter cooling: right ventricular free wall, septum, and left ventricular free wall temperatures were 7° ± 2°, 8° ± 2°, and 7° ± 2°C, respectively, after an initial 10 mL/kg of blood cardioplegia. Transatrial CS cardioplegia was used in 45 aortic valve replacements, 22 mitral valve replacements, 4 triple-valve replacements, 6 congenital lesions, and 148 coronary revascularizations, including 40 redo operations. Atheromatous material was routinely flushed retrogradely from cut old vein grafts during revascularization; 70 revascularizations (47%) were performed urgently for acute infarction or jeopardized myocardium. No heart block or CS injury occurred, and inotrope use dropped to 10% of patients (from 38% in the previous 256 patients with antegrade blood cardioplegia). We conclude that the CS can be cannulated transatrially and that CS cardioplegia is more facile than antegrade cardioplegia. Transatrial CS cardioplegia appears to be the cardioplegic method of choice.

(Ann Thorac Surg 1990;50:882–7)

The concept of providing nutrient flow or cardioplegic delivery to the myocardium through the coronary veins is not new [1–6]. Indeed, Lillehei and associates [7] successfully treated calcific aortic stenosis by means of a pump oxygenator and retrograde coronary sinus perfusion as early as 1955. The techniques of retrograde coronary sinus perfusion or cardioplegia delivery have changed little since that time. Heretofore, it has been necessary to provide two caval cannulas, snares around each cava during bypass, and an atriotomy and visual localization of the coronary sinus to deliver retrograde cardioplegia. Thus, despite any real or perceived advantages that retrograde cardioplegia might have over antegrade cardioplegia in terms of myocardial protection, difficulty in administering retrograde cardioplegia precludes its widespread use.

Beginning in February 1988, we began applying our laboratory-derived concept of transatrial cannulation of the coronary sinus [8, 9] to our clinical practice in an effort to make retrograde cardioplegia as facile as antegrade cardioplegia. This report details the first 225 patients treated with transatrial coronary sinus cardioplegia (TACSC).

Material and Methods

Patients

All patients receiving cardioplegic arrest on the service of the primary author (S.R.G.) from February 1988 to January 1989 at the University of Maryland Hospital, Baltimore, MD, and from February 1989 to May 1989 at Loma Linda University Medical Center, Loma Linda, CA, had transatrial cannulation of the coronary sinus as the sole form of cardioplegia delivery. Additionally, from March 1988 to May 1989, patients undergoing initially aortic valve replacement and progressing to redo coronary artery bypass operations and patients undergoing high-risk or emergent coronary artery bypass operations at the University of Maryland Hospital had TACSC as the sole method of cardioplegia delivery.

In all, a total of 225 patients ranging in age from 2 years to 89 years were treated with TACSC. Operations performed included 45 aortic valve replacements, 6 combined with coronary bypass, and 22 mitral valve replacements or repairs, 8 combined with coronary bypass; 6 of these operations were combined aortic and mitral valve procedures. Four patients underwent triple-valve repair and/or replacement. Six congenital lesions consisting of atrial or ventricular septal defects were treated. There were 148 coronary artery revascularizations, of which 40 were reoperations. Seventy patients were operated on emergently (within 6 hours of diagnosis) or urgently (within 24

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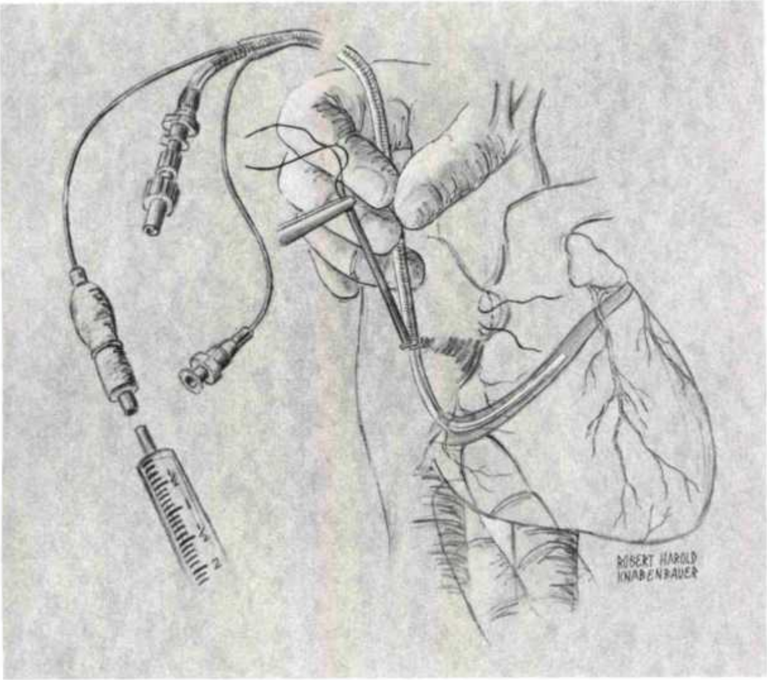

Fig 1. Assistant's-side insertion of Gundry RCSP catheter through pursestring high in right atrium. Catheter is shaped in hockey-stick configuration or gentle curve and passed into great cardiac vein, beyond the posterior interventricular vein. Note surgeon's left hand on diaphragmatic surface of the heart with index and long fingers at junction of inferior vena cava and atrioventricular groove, to guide catheter into coronary sinus.

hours) for unstable angina or acute infarction. On the primary author's service, where technically feasible, at least one internal mammary artery was used in the revascularizations.

Technique

Cannulation sutures are routinely placed in the aorta, atrium, and/or cavae for appropriate arterial and venous cannulation. An additional pursestring is placed just inferior to the junction of the right atrial appendage and right atrium (Figs 1, 2) for TACSC. After adequate heparinization, the aorta is cannulated for arterial inflow. A 180-cm (6-ft) arterial pressure line is passed over the ether screen to the anesthesiologist, who connects it to the pulmonary artery pressure transducer and flushes it with heparinized saline solution. This line will subsequently be connected to the distal pressure line incorporated into the TACSC catheter.

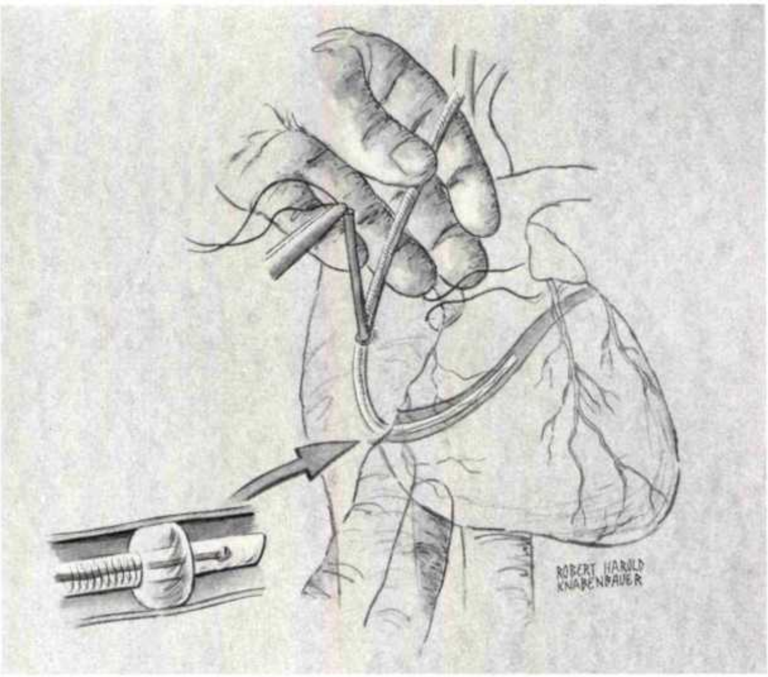

Fig 2. Surgeon's-side insertion of Gundry RCSP cathether through pursestring high in right atrium. Note insert showing balloon inflated to seal coronary vein during cardioplegia delivery. Balloon remains inflated until cross-clamp is removed. Distal pressure lumen is connected to transducer above ether screen.

TRAL
RARY

The Gundry RCSP catheter (DLP, Inc, Grand Rapids, MI) with its flexible stylet is shaped into a hockey-stick configuration with a gentle curve on its end (see Figs 1, 2). If patient stability permits, a stab wound is placed inside the TACSC pursestring, the RCSP catheter is placed into the right atrium, and the snare is gently snugged. The catheter can be introduced from either the operator's or first assistant's side of the table. We and others have found that when first learning the technique, insertion from the assistant's side is inherently easier (see Fig 1), and it is explained first.

The catheter is held in the right hand, and the left hand index and long fingers are placed on the diaphragmatic surface of the heart at the junction of the inferior vena cava and atrioventricular groove. The tip of the catheter is located with these fingers by feeling through the atrial wall. A gently pushing motion with the right hand is used to guide the tip of the catheter into the coronary sinus and advance it beyond the posterior interventricular vein upward toward the left shoulder. The catheter's advancement is confirmed by palpation with the fingers of the left hand.

Intubation from the surgeon's side is somewhat more difficult at first, as the angles used for both hands limit motion. The catheter is grasped with the left hand while the right hand is placed along the diaphragmatic surface of the heart (see Fig 2). Either the index and long fingers or the little and fourth fingers at the inferior vena cava and atrioventricular groove junction are used to locate the catheter tip transatrially and guide it into the coronary sinus and into the great cardiac vein. From the surgeon's side, the cannula is more likely to either enter the right ventricle or get caught in the posterior interventricular vein but these are easily recognized and corrected.

Confirmation of placement is obtained by manually palpating the catheter in the atrioventricular groove posteriorly. If not felt, it is most likely in the right ventricle. We have not seen arrhythmias once the catheter is in the great cardiac vein. Arrhythmias usually imply that the catheter is within the right ventricle. Further confirmation of placement is done by withdrawing the stylet and observing black pulsatile blood in the cannula. The cannula is secured by snaring down the snugger and securing with a free tie around the movable ring on the cannula. The previously flushed 180-cm (6-ft) pressure line is connected to the clear pressure port on the RCSP cannula and a coronary sinus waveform tracing is observed on the monitor.

Once in place, venous cannulas are inserted per the surgeon's preference. Remarkably, there have been no catheter dislodgements by the venous cannulas. Bypass is commenced in standard fashion. If patient instability warrants placement on bypass before insertion of the RCSP cannula, the cannula can be placed during bypass. Usually, it is easier to intubate the coronary sinus by passing the RCSP cannula over the venous cannula, but occasionally intubation is only possible behind and under the venous cannula.

Administration of Cardioplegia

In this series, all patients were cooled to a core temperature of 26° to 28°C. A cardiac insulator pad was placed behind the heart. Cold blood cardioplegia was used as the only cardioplegia in all patients.

During cooling, the blood cardioplegia line from the perfusionists was flushed and connected to the central RCSP catheter port. A 3-mL syringe was attached to the blue balloon port. Initially, a left ventricular vent was placed through the right superior pulmonary vein in all patients, but in the last 100 patients, only an aortic needle vent has been used for revascularizations.

When the patient was systemically cool, or when fibrillation occurred, iced saline solution or Ringer's lactate slush was applied to the heart, an aortic cross-clamp applied, and retrograde cardioplegia started at a flow of 100 mL/min. Three milliliters of air was put into the balloon of the RCSP catheter and the anterior heart inspected. The veins were noted to be full and red and the arteries blue! Initial suction on the aortic needle vent also revealed blue, deoxygenated blood. Simultaneously, the coronary sinus pressure was observed on the monitor. The pressure usually rose to 30 to 50 mm Hg as the perfusionist increased flow to 250 to 300 mL/min. If the pressure did not rise, either the balloon would be inflated further, or the catheter had dislodged. We have used up to 5 mL of air without problems.

Cardiac arrest usually occurred within 20 to 30 seconds. Occasionally, fine fibrillation was seen in the most superior aspect of the right ventricular outflow tract. This ceased quickly. A total dose of 10 mL/kg was given as an initial bolus. Repeat 5-mL/kg doses were given every 30 minutes or whenever myocardial temperatures exceeded 20°C.

All distal vein graft anastomoses were completed first. Redo operations were performed by cutting old patent grafts in half before starting retrograde cardioplegia. In most cases, proximal anastomoses were performed during rewarming, using a partial occluding aortic clamp. Once the aortic cross-clamp was released, the balloon on the RCSP catheter was deflated, but the catheter was left in place until all anastomoses were complete.

Results

The coronary sinus was intubated in all cases. There were 6 cases of catheter dislodgement, all but 1 case occurring during a single week when perfusionists used up to 500 mL/min flow through the catheter, causing a "garden hose" effect. All catheters were easily replaced by reinserting the stylet.

There was no coronary sinus or cardiac vein damage or disruption. There was no evidence of atrioventricular groove hemorrhage or myocardial hemorrhage. Coronary sinus pressures never exceeded 60 mm Hg, although when manipulating the heart, the pressure monitoring port could be compressed intermittently, simulating higher pressures.

Myocardial temperatures were recorded in the right ventricular free wall, septum, and left ventricular free wall in the first 100 patients. Temperatures after the initial dose of 10 mL/kg of cardioplegia were as follows: right ventricle, 7° ± 2°C; septum, 8° ± 2°C; left ventricle, 7° ± 2°C. No temperature differences were found between patients with patent or acutely occluded arteries. Due to this uniformity of cooling, temperature monitoring is not a routine part of our current practice.

Inotropes were used in 23 of 225 patients, usually low doses (2 to 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$) of dopamine or dobutamine. This compares with 97 of 256 (38%) patients receiving antegrade blood cardioplegia in the same practice during 1987 [10]. There were two deaths at Loma Linda, one occurring in a balloon pump–dependent patient with an ejection fraction of less than 0.20 arriving in shock, the second occurring in a bedridden 79-year-old woman who survived a redo double-valve replacement and triple coronary artery bypass but died of hospital-acquired pneumonia 2 weeks postoperatively.

Intraoperative echocardiography of prebypass and postbypass function was obtained using transesophageal probes in valve and valve/revascularization procedures. All 40 patients studied had preserved or improved right ventricular function before chest closure.

Comment

Retrograde cardioplegic protection of the myocardium has entered a period of rapidly increasing interest by researchers and clinicians. Its appeal stems from the known limitations of antegrade delivery of cardioplegia, namely, inadequate distribution of cardioplegia distal to coronary artery lesions [11], damage to coronary ostia, difficulty in ostial cannulation, and distal emboli from previous vein grafts.

In contrast, retrograde cardioplegia provides uniform distribution of cooling throughout both ventricles [8, 9] despite coronary lesions (Table 1). Coronary ostia are neither sought nor injured with retrograde methods. Old vein grafts can be cut and any distal embolic material flushed retrogradely out of the coronary arteries and old grafts. Moreover, reoperations with patent internal mammary arteries are possible as all parts of the heart receive adequate myocardial protection. Mitral and aortic valve operations are accomplished more expeditiously; the operation can proceed without the interruption of cannulating coronary arteries or putting retractors down to restore aortic valve competence before more antegrade cardioplegia. Finally, myocardial protection in acute aortic dissection is equally enhanced.

Despite these obvious benefits, the principal limitation of retrograde cardioplegia has heretofore been the complicated steps required to cannulate the coronary sinus, ie, bicaval cannulation, caval snares, and an atriotomy. Not only are these time consuming, but valuable cross-clamp time can be expended while searching for and then cannulating the coronary sinus through a small atriotomy. These drawbacks notwithstanding, Menasché and associates [12] have reported on 500 consecutive aortic valve replacements using retrograde coronary sinus cardioplegia with results at least equal to those of antegrade cardioplegia.

Table 1. Observed Benefits of Transatrial Coronary Sinus Retrograde Cardioplegia

Operation	Benefits
Aortic valve replacement	No searching for coronary ostia
	Exploration and excision of aortic valve while giving dose
	Working on valve while giving initial and repeat cardioplegia
	Flushing calcific debris from coronary ostia
	Final dose can "clean" coronary arteries and root
Mitral valve replacement	Exploration and retraction of atrium while giving initial dose
	Continuing the operation in the atrium while giving subsequent doses
Coronary revascularization	Even cooling of entire heart
	Elevation of the heart and dissection of coronary arteries while giving cardioplegia
	Protection of myocardium in areas destined for IMA graft(s)
	Cooling protection of myocardium distal to acute occlusions
Redo revascularization	Prevention of distal vein graft atheromatous emboli
	Flushing atheromatous emboli out of coronary arteries
	Protection and cooling of heart with intact IMA grafts

IMA = internal mammary artery.

It is our belief that retrograde cardioplegia must be as facile to administer as antegrade cardioplegia for it to become more widely used. To that end, we have now demonstrated that the coronary sinus can be easily consecutively intubated transatrially by a variety of surgeons and used with good results on a variety of cardiac lesions. In doing so, we have become convinced that in certain lesions, transatrial coronary sinus cardioplegia is more facile than antegrade cardioplegia. These lesions include all valve replacements, valve and coronary combination operations, redo operations, and revascularizations for acute myocardial ischemia. With practice, intubating the coronary sinus is as easy as putting a cardioplegia needle into the ascending aorta.

We have further demonstrated that good clinical results can be obtained without combining retrograde with prior doses of antegrade cardioplegia. This "antegrade retrograde" method unnecessarily complicates a simple procedure. Moreover, this practice may prove harmful if valuable time is lost searching for coronary ostia, if coronary ostia are damaged, or if vein graft atheromatous material is flushed down into coronary arteries antegradely.

Two additional findings require comment. First, we have used pressures as great as to 60 mm Hg within the coronary sinus without damage. Indeed, research on the beating, working heart with an arterialized, occluded coronary sinus found that pressures as great as 60 mm Hg could be tolerated without hemorrhage or edema [13]. By decompressing the nonworking heart, as in this study, high pressures may be tolerated even further.

Finally, a key to facile retrograde cardioplegia is the placement of the balloon and catheter well into the great cardiac vein, usually past the posterior interventricular vein. Experimental results of such placement have previously confirmed the excellent cooling of the entire heart by this method [8, 9]. Clinical temperature data in this report further support the more distal placement of the catheter to avoid dislodgement during cardiac manipulation.

Based on our experience with 225 patients, we conclude that the coronary sinus can be easily intubated transatrially using a catheter with a flexible stylet. The use of surgeon-controlled balloon inflation and distal pressure monitoring have ensured adequate delivery of cardioplegia without coronary sinus or myocardial injury. Transatrial coronary sinus cardioplegia may be the cardioplegia method of choice for all operations, but at the very least it offers a superb alternative method of cardioplegia delivery for valve operations, aortic root reconstructions, revascularizations, and acute myocardial ischemia.

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DISCUSSION

DR RICHARD E. CLARK (Bethesda, MD): I have had the privilege of hearing some of Dr Gundry's work before when he was a visitor at the National Institutes of Health. I think this work is important. I have a couple of questions for you with regard to it.

Why have you abandoned use of stopping the heart immediately with an antegrade dose to give you additional time to put in the retrograde? Second, you noted that there was a difference in inotrope use and you have disclaimed any efficacy there. For your patient population, particularly in those undergoing redo operations, have you found any difference in mortality, which you carefully avoided mentioning?

DR JOSEPH M. CRAVER (Atlanta, GA): I just have two technical questions. One relates to the particular benefit of retrograde cardioplegia in those patients who have chronic total occlusion of the proximal right coronary artery in protecting the right ventricle. In this circumstance antegrade cardioplegia administration may leave this area, which is also subject to surface warming, unprotected and in jeopardy of further dysfunction. With regard to that, how do you prevent an insertion of the catheter too deeply into the coronary sinus whereby the retrograde flow may not perfuse the inferior vein and the other right-sided structures? Second, how do you carefully administer the amount of fluid into the balloon of the catheter to avoid any local trauma to the area of the coronary sinus where the balloon might be located?

DR KIT V. AROM (Minneapolis, MN): I have one brief question. I may have misunderstood. Do you run cardioplegia for 300 mL/min continuously?

DR GUNDRY: Thank you very much for those very pertinent remarks. First of all, one of the reasons we chose to do this study is to perhaps demonstrate to ourselves and to a large number of people that there is no need, at least in our hands, for any combination of antegrade and retrograde delivery of cardioplegia. We believe that the initial use of antegrade cardioplegia just complicates an extremely simple technique, and have always been taught that the simpler, the better. Antegrade cardioplegia would not be efficacious in those patients who have aortic insufficiency, and particularly in redo operations where our main fear is delivering atheromatous material down vein grafts. The antegrade cardioplegia is not needed in our hands. We get immediate cessation of function in the heart using retrograde cardioplegia, and I believe many reports about retrograde cardioplegia being very slow to stop the heart have been a result of a fear of, in a beating heart, going above the magic pressure of 60 mm Hg.

I may just say that the coronary veins have no capillaries, so that when you blow up the coronary sinus balloon you, in a sense, have a capillary wedge pressure effect. And if the ventricle is not completely decompressed, you will actually view the left ventricular pressure through the coronary veins. The coronary veins during systole are frequently exposed to left ventricular pressure, and as long as you have egress of blood through some vent somewhere, you can exceed this pressure. So we have not found any need for antegrade cardioplegia. If people want to use that, that is just fine. There are plenty of systems on the market. I just feel it is a little too complex.

In regard to our mortality statistics, there were a total of six deaths in this group. All of those deaths except one occurred in patients on intraaortic balloon pumps directly from the catheterization laboratory, two of them subsequently needing left and right ventricular assist device support. Surprisingly, the rest of the patients undergoing emergent revascularization emerged from the operating room usually not requiring any inotropes, which, again, has been a pleasant surprise to all of us using this technique.

With regard to the question about putting the cannula too far, I believe this is an unfortunate misconception that has been perpetrated through the years. Because of the dense venous network and venous arterial connections within the heart, you do not have to worry about the right ventricular posterior branches with your coronary sinus cannula. We have always put this cannula beyond the posterior interventricular veins so that it will not come out, and we are delighted see our cardioplegia moving through these veins, even using the veins as an egress of the heart. And as you can see from our temperature mapping, the right ventricle is extremely well cooled.

With regard to pushing the catheter too far and making the balloon too big, the beauty of this catheter design is that if the balloon encounters any resistance to its inflation, the additional air pressure will pop off into the pressure pop-off device built into the catheter. And I am pleased to report that there has yet to be a perforation of a coronary sinus using this particular catheter. Even my residents can put this in and not do any harm.

Finally, as to the amount of cardioplegia, we use a bolus technique of cardioplegia, not continuous. We find the bolus technique much easier to use. We have tried continuous retrograde cardioplegia but, unfortunately, you have to use snares around the coronary arteries because the coronary arteries, of course, are the egress for some of this cardioplegia, and you have a constant little drip bothering you. So this report details intermittent doses of cardioplegia.

Continuous Epidural Hydromorphone for Postthoracotomy Pain Relief

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Forty-four patients were treated with a continuous infusion of lumbar epidural hydromorphone (0.05%) after thoracic operations. Postoperatively, visual analog pain scores were obtained. On postoperative day 1 and 2, more than 90% of the patients experienced either no pain (visual analog pain scale = 0) or mild pain (visual analog pain score = 1 to 3) at rest. The incidence of side effects (hypoventilation, pruritis, and nausea) was less than reported with other epidurally administered opioids. Continuous infusion of lumbar epidural hydromorphone produced safe, predictable analgesia after thoracotomy.

(Ann Thorac Surg 1990;50:888–93)

Opioids administered by the epidural route relieve postthoracotomy pain while preserving pulmonary function [1]. The most frequently used agent, morphine, has been associated with delayed respiratory depression [2]. In an effort to minimize the incidence and severity of this and other side effects, alternative agents have been tried [3–5]. We previously reported that lumbar epidural hydromorphone (Dilaudid) produced excellent analgesia after a pulmonary operation when administered by intermittent bolus [3]. This report describes our experience with continuous infusion of lumbar epidural hydromorphone for postthoracotomy analgesia.

Material and Methods

With the approval of the Human Subjects Committee of Stanford University Medical Center, 44 patients undergoing major noncardiac thoracic operations were studied prospectively during a 12-month period (Table 1).

For editorial comment, see page 862.

On the patient's arrival in the operating room, a catheter was placed in the L2–3 or L3–4 epidural space and advanced 3 to 5 cm cephalad. After administration of a test dose of 3 to 4 mL of 1.5% lidocaine with epinephrine (1/200,000), 12 to 18 mL of either 2% lidocaine or 0.5% bupivacaine was administered through the epidural catheter. After correct epidural placement was confirmed by demonstration of bilateral sensory block, general anesthesia was induced with thiopental. Muscle relaxants were administered to facilitate airway intubation. Anesthesia was maintained with isoflurane (0.8% to 1.5%) and oxygen. Local anesthetics were administered through the epidural catheter during the operation to supplement general anesthesia. No parenteral opioids or sedatives were administered either preoperatively or intraoperatively. Additional muscle relaxants were used if needed. Fluids were intentionally restricted in this group of patients; if hypotension occurred, it was treated with ephedrine (5 to 25 mg intravenously [IV]) in divided doses.

Approximately 1 to 2 hours before completion of the operation a single or combined dose of opioid(s) was injected as a bolus through the epidural catheter (Table 2).

All patients were extubated in the operating room as soon as spontaneous breathing was established, usually within 10 minutes after completion of the operation. Patients were then taken to the intensive care unit (ICU) (39 patients) or to the postanesthesia care unit (5 patients) depending on the severity of their pulmonary disease and other diseases, the extent of operation, or both. Patients in the postanesthesia care unit were transferred to a regular surgical unit within 2 hours.

Each patient was interviewed by the trained nurse from our acute pain service 30 minutes after arrival in the ICU or postanesthesia care unit. Patients were asked to score their pain by using a visual analog scale. A 10-cm line with the words "no pain" at one end and "worst pain imaginable" at the other end was presented to each patient. Patients placed a mark along the 10-cm line to indicate the amount of pain they were experiencing at that particular moment. The distance from the end of the line marked no pain to the patient's mark was measured and recorded. The result is an analog measurement of pain intensity from 0 (no pain) to 10 (worst pain imaginable). Such visual analog pain scores (VAPSs) were obtained and recorded with the patient at rest ($VAPS_{rest}$) and after cough or movement ($VAPS_{mvmnt}$) [6]. Pain scores were categorized into one of four groups: no pain (VAPS = 0), mild pain (VAPS = 1 to 3), moderate pain (VAPS = 4 to 7), and severe pain (VAPS = 8 to 10). Pain relief was considered good with scores of 0 to 3, fair with scores between 4 and 7, and poor with scores between 8 and 10.

A continuous epidural infusion of hydromorphone (0.3 mg/h) in preservative-free saline solution (0.05% solution)

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Table 1. Surgical Procedures for Which Continuous Infusion of Lumbar Epidural Hydromorphone Was Used to Treat Postoperative Pain

Procedure	No. of Patients[a]
Posterolateral thoracotomy	
Lobectomy	16
Wedge resection	4
Pneumonectomy	4
Bleb resection	3
Tumor debulking	3
Exploratory	3
Chest wall resection	2
Drain effusion	1
Evacuate clots	1
Sternotomy	
Thymectomy	3
Lobectomy	2
Bilateral wedge resections	1
Excision of mediastinal mass	1
Total	44

[a] M = 27, average age 56.7 years; F = 17, average age 47.3 years.

was then initiated. If a patient complained of pain, epidural fentanyl (50 to 75 μg) in 15 to 20 mL of preservative-free saline solution was administered as a bolus.

Table 2. Average Pain Scores (Range) in the ICU or PACU on Day of Operation (POD 0)

Intraoperative Opioid(s)[a]	No. of Patients	$VAPS_{rest}$
Hydromorphone (mg)		
1	14	3.6 (0–10)
1.5	3	3.6 (0–9)
2.0	1	8.0
Hydromorphone (mg)/fentanyl (μg)		
0.75/100	2	5.0 (1–9)
1/50	4	3.2 (0–8)
1/75	2	4.5 (4–5)
1/100	3	1.3 (0–3)
Hydromorphone (mg)/morphine(mg)		
0.75/5	9	3.5 (0–6)
1/5	2	7.0 (7.0)
Fentanyl (μg)		
100–200	3	2.3 (0–7)
Morphine (mg)		
4	1	0

[a] Patients received either a single opioid or a combination of two drugs intraoperatively.

ICU = intensive care unit; PACU = postanesthesia care unit; POD 0 = awakening from anesthesia (postoperative); $VAPS_{rest}$ = visual analog pain scores at rest.

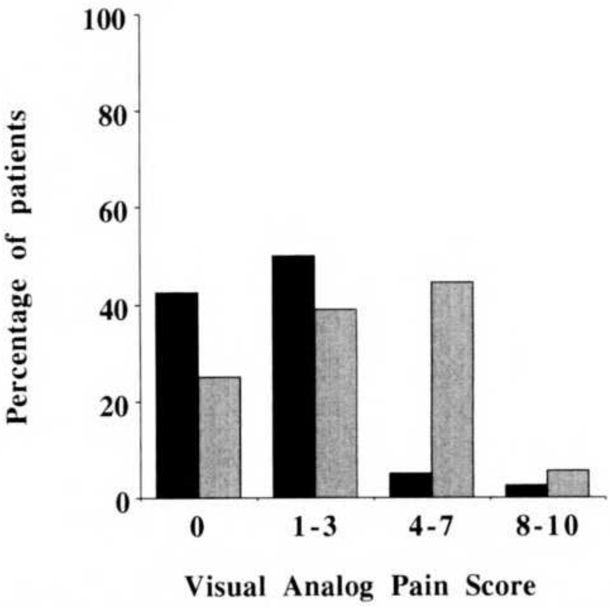

*Fig 1. Visual analog pain score (VAPS) at rest (*solid bars*) and with movement (*gray bars*) were categorized into one of four groups: no pain (VAPS = 0), mild pain (VAPS = 1 to 3), moderate pain (VAPS = 4 to 7), and severe pain (VAPS = 8 to 10). The percentages of patients in each pain score group on postoperative day 1 are shown. After thoracotomy, more than 90% of patients had no pain or mild pain at rest while receiving continuous infusion of lumbar epidural hydromorphone.*

Each patient was visited by the acute pain service team twice a day. Visual analog pain score at rest and with movement, incidents of breakthrough pain, and treatment received were recorded. Complications and side effects were assigned a score (mild, moderate, severe) and treated with nalbuphine (pruritis), metoclopramide (nausea), midazolam (anxiety), or naloxone (sedation or hypoventilation).

Under supervision of the surgical team, the epidural catheter was left in place until the patient was able to tolerate oral analgesics, at which time the catheter was removed. Patients were then asked if they had been satisfied with their analgesia and whether they would choose epidural opioids again.

Results

No intraoperative complications were attributable to either the regional or general anesthesia. The initial pain scores on awakening from anesthesia (POD 0) are shown in Table 2. No single drug or combination of opioids appeared to be superior, but groups were too small to allow statistical intergroup comparison.

Figures 1 and 2 summarize the pain scores on the morning of the first (POD 1) and second (POD 2) postoperative days after at least 16 hours of continuous hydromorphone infusion. The percentage of patients in each of the four pain groups is shown.

On the day of operation (POD 0), 68.2% of patients required between one and four boluses of epidural fentanyl for additional pain relief. By the following morning

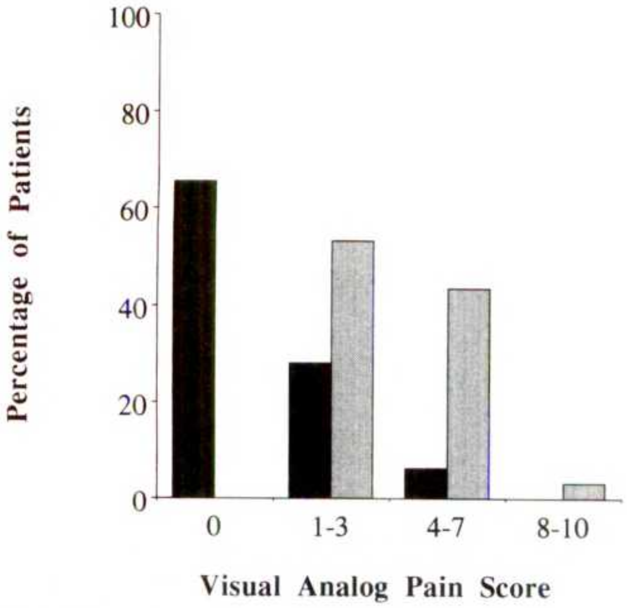

Fig 2. The percentages of patients in each pain group on postoperative day 2 are shown. Visual analog pain scores at rest (solid bars) *and with movement* (gray bars) *were measured. Again, more than 90% of patients had no pain or mild pain at rest while receiving continuous infusion of lumbar epidural hydromorphone.*

(POD 1) and thereafter during the time the hydromorphone infusion was continued (1 to 5 days), only 12 patients (27.3%) required one to four additional interventions with supplemental analgesia, usually for increased pain during ambulation or chest physiotherapy (Table 3).

Complications associated with continuous epidural hydromorphone are shown in Table 4. Because all patients had indwelling urinary catheters while receiving epidural medication, urinary retention was not a problem. Six patients received naloxone for hypoventilation associated with sedation. In 4, the hypoventilation was mild (arterial carbon dioxide tension < 52 mm Hg), and all were easily arousable by verbal stimuli. The epidural infusion was decreased to a level of 0.1 to 0.2 mg/h, and hypoventilation did not recur.

Three patients (6.6%) required reintubation. One had mild carbon dioxide retention (arterial carbon dioxide tension < 52 mm Hg). After treatment with naloxone, 0.4 mg IV in divided doses, he was extubated 2 hours later. The second patient was an elderly man who underwent pneumonectomy. Although comfortable, he was very anxious postoperatively and received midazolam, 6 mg IV. After this heavy sedation, he required ventilatory support for several days but recovered uneventfully. Three other patients also received midazolam for anxiety but did not require ventilatory assistance. The third patient became septic and hemodynamically unstable several days after operation and required ventilation at that time.

Table 3. Number of Patients Requiring Supplemental Epidural Fentanyl (50–75 μg) Bolus Intervention From Postoperative Day 1 On

No. of Patients (n = 43)[a]	No. of Fentanyl Boluses
31	0
6	1
1	2
4	3
1	4

[a] One patient had the catheter removed on the night of postoperative day 0.

Table 4. Side Effects Associated with Continuous Lumbar Epidural Hydromorphone Expressed as Percentage of Patients

Degree	Pruritis	Nausea	Anxiety	Hypoventilation
Mild	11.4	4.5	0	9.0
Moderate	18.2	9.0	6.8	4.4
Severe	0	0	2.2	0
Total	29.6	13.5	9.0	13.4

Most patients remained in the ICU only overnight and were transferred to regular surgical wards on the morning after operation. The epidural catheters remained in place for an average of 2.6 days (range, 1 to 5 days). In most patients, the catheters were removed when oral analgesics were sufficient for pain relief. In 6 patients, the catheters were removed for other reasons (Table 5).

All but 2 (4.5%) patients were satisfied with the epidural hydromorphone and stated they would choose it again. One patient with mild discomfort ($VAPS_{rest} = 3$, $VAPS_{mvmnt} = 4$) preferred IV opioid analgesia. The other dissatisfied patient had excellent pain relief ($VAPS_{rest} = 0$) but experienced pruritis and requested that the catheter infusion be stopped on POD 1. The pruritis resolved after discontinuation of the epidural hydromorphone.

Comment

Severe postthoracotomy pain interferes with ambulation, deep breathing, and coughing, thus increasing the risk of pulmonary complications (atelectasis, pneumonia, pulmonary emboli).

Numerous regimens have been used to treat thoracot-

Table 5. Reasons for Discontinuation of Epidural Catheter[a]

Reason	No. of Patients
Patient able to tolerate oral analgesics	38
Catheter contaminated	1
Catheter leaking	1
Catheter fell out	1
Patient febrile	2
Patient complained of pruritis	1

[a] Average duration of continuous infusion of lumbar epidural hydromorphone was 2.6 days (range, 1 to 5 days).

omy pain [7]. Parenteral opioids have been given on demand or by patient-controlled analgesia, and local anesthetics have been used with intercostal blocks, either intrathecally or epidurally, or both, and more recently through intrapleural catheters.

During the past decade, "spinal" opioids have been used [2, 8]. Intrathecal or epidural opioids provide excellent pain relief, preserve pulmonary function, and are free of many of the complications associated with local anesthetics and parenteral opioids [2].

When we first began using epidural opioids for postoperative analgesia, we were concerned about the potential risks of delayed respiratory arrest. Between 1983 and 1986, all surgical patients, including those undergoing thoracotomy, were allowed epidural opioids only while in the ICU. During that 4-year period, no instances of apnea were related to this therapy. Thereafter, as a trial, for 1 year surgical patients were allowed to receive epidural opioids on a single selected surgical ward staffed by specially trained nurses. No patient experienced clinically significant respiratory depression. Beginning in 1988, patients receiving epidural opioids have been hospitalized on all surgical wards at Stanford University Hospital. Continued nursing education, standard orders, hospital protocols, and medical supervision by physicians familiar with the actions and side effects of epidural opioids has allowed patients to be treated safely with epidural opioids outside the ICU. Copies of these protocols and instructions are available from the Department of Nursing at our hospital.

Since January 1988, our acute pain service staff members have prospectively followed more than 1,500 of these patients and have not observed a single episode of apnea related to epidural hydromorphone. In fact, our younger thoracotomy patients go directly from the postanesthesia care unit to a regular surgical ward without being admitted to the ICU. In the absence of surgical complications, most patients admitted to the ICU are transferred to a regular surgical ward on the first postoperative day, where they continue to be treated with epidural opioids.

We previously reported our experience with intermittent bolus epidural hydromorphone for postthoracotomy analgesia [3]. We believe that hydromorphone is superior to epidural morphine because it produces excellent analgesia with fewer side effects [3]. Commercially available hydromorphone is also inexpensive and contains no preservatives. After the change in our hospital practice allowing epidural drugs outside the ICU, we began administering epidural hydromorphone by continuous infusion [9]. Because catheters can be kept in place for several days, intermittent delivery of drugs is neither practical nor necessary. Bolus administration of hydromorphone produces excellent pain relief after thoracotomy; however, delivering opioid by continuous infusion allows achievement of a more consistent level of analgesia without delays caused by waiting for medical or nursing personnel to "top-up" the epidural catheter. Continuous infusion of opioid is a more advantageous means of providing pain relief.

In our series, initial $VAPS_{rest}$ on awakening from general anesthesia ranged from no pain (0) to very severe pain (10). In the immediate postoperative period, some patients still had a sensory block to pain from epidural local anesthetics whereas others did not. We could not determine which intraoperative drug or combination worked best because groups were too small for comparisons.

The most reliable indicators of postthoracotomy analgesia are pain scores after the patient has fully recovered from anesthesia. On POD 1, 92.5% of our patients either had no pain ($VAPS_{rest}$ = 0) or mild discomfort ($VAPS_{rest}$ = 1 to 3). On POD 2, 93.7% of patients were comfortable at rest. Although a larger number of patients had moderate pain (VAPS 4 to 7) with movement, this pain was transient, usually lasting only a few seconds after the initial stimulus. If the pain persisted, a bolus of epidural fentanyl was given. Only 27.3% of patients requested this additional pain medication, attesting to the efficacy of the epidural hydromorphone infusion.

Asantila and co-workers [10] administered 4- or 6-mg boluses of morphine through thoracic epidural catheters for postthoracotomy pain. At 24 hours (equivalent to our POD 1), average VAPSs were 4.1 and 2.7, respectively. All of their patients required supplementary analgesics throughout their recovery.

El-Baz and colleagues [11], using a 10-cm visual analog pain scale identical to the one we used, administered morphine through thoracic epidural catheters either as on-demand intermittent bolus (5 mg) or by continuous infusion (0.1 mg/h). On the first day after thoracotomy (POD 1), patients receiving bolus morphine had a mean VAPS of 2.1 and those receiving continuous morphine infusion had a VAPS of 1.9. When the percentage of patients with satisfactory pain relief (VAPS 0 to 3) is considered, only 70% of the intermittent morphine group and 65% of the continuous morphine infusion group had a VAPS of 0 to 3 (11). The remaining patients had poor or no pain relief. Neither bolus nor continuous-infusion epidural morphine provided consistent analgesia. More than 90% of patients receiving continuous hydromorphone in our series had a VAPS of 0 to 3.

The patients of El-Baz and colleagues received 0.1 mg/h of epidural morphine, whereas our patients received 0.3 mg/h of hydromorphone. We were administering larger amounts of a more potent drug, albeit through a more distant lumbar rather than thoracic catheter. This may account for the superior analgesia in our patients. Perhaps if the concentration of morphine were increased, similar levels of pain relief could be achieved with morphine; however, increasing the dose of thoracic epidural morphine probably increases the incidence of side effects, particularly respiratory depression [3].

Occasional episodes of increased pain occur after thoracotomy, particularly during active periods of ambulation or chest physiotherapy. Only 12 patients in our series required any additional analgesia after POD 0; of these, half needed only a single bolus of epidural fentanyl. No patient required more than four interventions. All El-Baz's patients required supplementation with morphine (2 mg/bolus IV) throughout the entire 72-hour study. In their intermittent bolus epidural morphine group, the average requirement was 15.9 mg (eight injections) of supplemental IV morphine on POD 1 alone. The continuous infusion group received 5.8 mg (three injections) of IV morphine on POD 1.

We prefer epidural fentanyl to other opioids for breakthrough pain because this highly lipid-soluble agent has a rapid onset of action. Improved analgesia is usually noted within 5 minutes after its administration.

Because epidural morphine produces the longest duration of analgesia of the opioids, it may be indicated when a single injection or intermittent bolus technique is used. Hydrophilic morphine remains in the cerebrospinal fluid, however, and eventually may reach the brain in concentrations great enough to cause respiratory depression many hours after administration [2]. Lipophilic opioids with higher spinal cord receptor affinity and lower cerebrospinal fluid concentrations are believed to have fewer delayed central respiratory effects [2]. Therefore, shorter acting, lipophilic opioids (fentanyl, sufentanil) have also been used for postthoracotomy pain relief.

With continuous thoracic epidural infusion of fentanyl ($0.6\ \mu g \cdot kg^{-1} \cdot h^{-1}$) average VAPSs on POD 1 and 2 were between 2 and 3, but more than 65% of patients required one to three supplemental boluses of epidural fentanyl for pain relief [5]. A very high incidence of drowsiness (78%), pruritis (44%), and nausea (22%) was reported.

After thoracotomy, lumbar epidural sufentanil (30, 50, or 70 μg) produced a VAPS of less than 2 within 30 minutes, but an unusually large number of patients had to be withdrawn from this study because of life-threatening acute respiratory depression [4]. Similar findings, including respiratory arrest after 50 μg of epidural sufentanil have been reported by other investigators [12].

Even when opioids are introduced into the epidural space, high blood levels occur. Epidural morphine is rapidly absorbed by the epidural venous plexus, resulting in a serum drug level almost equivalent to that achieved with an identical IV dose [13]. Even higher blood levels have been demonstrated with epidural fentanyl and sufentanil [4, 14]. The spinal analgesic effects last long after the opioid has disappeared from the blood, but some of the immediate side effects (sedation, nausea, early respiratory depression) may not be spinal effects; instead they may be entirely due to high systemic levels. Such effects (drowsiness, acute respiratory depression) occur more often with the more lipophilic agents (fentanyl, sufentanil) [14, 15].

Although the hydrophilicity of hydromorphone is similar to that of morphine, almost no reports describe delayed respiratory depression after administration of epidural hydromorphone. To date, only one case report has described delayed respiratory depression from epidural hydromorphone in an elderly patient who received a very large amount of drug through a thoracic catheter after heavy premedication [16]. In our series, other side effects (sedation, nausea, pruritis) occurred less frequently than has been reported with more lipophilic agents.

Epidural opioids can be injected at either the lumbar or thoracic spinal level for treatment of postthoracotomy pain. A higher incidence of respiratory depression is reported when the thoracic route is used [3]. Local anesthetics and spinal opioids both provide excellent analgesia for thoracotomy incisions even when administered at the lumbar level, and because the risk of spinal cord damage is virtually eliminated when the lumbar route is chosen we prefer the lumbar epidural route for our thoracotomy patients.

In summary, continuous lumbar epidural hydromorphone (0.3 mg/h) produced predictable, safe analgesia. More than 90% of our patients experienced no pain or mild pain at rest. The incidence of drowsiness and sedation, and other complications (pruritus and nausea), was relatively low as compared with those occurring with other epidural opioids. Administration of lumbar epidural hydromorphone did not prolong the ICU stay of our patients. In fact, of the patients admitted to the ICU, most were transferred to a regular surgical ward on the day after operation. Epidural opioid analgesia allows earlier ambulation and cooperation in chest physiotherapy, which in turn reduces morbidity and shortens hospital stay. In the absence of surgical complications, the average length of hospitalization of our patients is 5 to 6 days. We recommend continuous infusion of lumbar epidural hydromorphone for postthoracotomy analgesia.

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Cardiac Transplantation for Hypoplastic Left Heart Syndrome: A Modified Technique

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A modified technique of infant orthotopic cardiac transplantation with arch reconstruction using bicaval cannulation is described, and the results in 4 infants with hypoplastic left heart syndrome are presented. This technique minimizes donor myocardial ischemic time and recipient circulatory arrest time.

(Ann Thorac Surg 1990;50:894–8)

Infant orthotopic cardiac transplantation for hypoplastic left heart syndrome has resulted in excellent short-term and midterm results [1, 2]. Technical advances [3], myocardial preservation techniques [4, 5], and improved immunosuppression regimens [6] have had a major impact on patient survival. Two technical factors that we think are important for operative success are total donor myocardial ischemic time and recipient circulatory arrest. The purpose of this report is to introduce a modified technique of infant orthotopic cardiac transplantation and extensive arch reconstruction for hypoplastic left heart syndrome that will decrease both donor myocardial ischemic and recipient circulatory arrest times.

For editorial comment, see page 864.

Material and Methods

Four patients with type I* hypoplastic left heart syndrome underwent orthotopic cardiac transplantation at age 9, 17, 22, and 39 days. Cardiopulmonary bypass and profound hypothermia (20°C) were established by pulmonary artery cannulation (8F arterial cannula; DLP Inc, Grand Rapids, MI) and bicaval venous cannulation (12F, superior vena cava; 16F, inferior vena cava) (Fig 1). Flow to the lungs is prevented by snares around the right and left pulmonary arteries. A cross-clamp is placed across the pulmonary artery upstream from the arterial cannula and just above the pulmonary valve. A ligature is placed around the ascending aorta and tied. The heart is excised with cardiopulmonary bypass continuing while the donor heart is arriving at the hospital (Fig 2). The left atrial donor–recipient anastomosis is completed while extracorporeal circulation is maintained. The donor arch is trimmed to size for anastomosis (Fig 3). A left ventricular cannula is placed through the left atrial appendage for instillation of cold saline solution.

Circulatory arrest is now initiated after the head vessels have been controlled by temporary occlusion. The ductus arteriosus is ligated and divided, and the main pulmonary artery is transected just proximal to the bifurcation of the right and left pulmonary arteries. The recipient aortic arch is prepared by aortotomy across the transverse arch and coarctation site (Fig 4). Once the aortic arch reconstruction has been completed and maneuvers to remove air have been performed, aortic cannulation is accomplished and cardiopulmonary bypass reinstituted (Fig 5). At this point, the cannula in the left atrium previously used to instill cold saline solution is converted to a vent to decompress the left ventricle. The right atrial and pulmonary artery anastomoses are performed with the heart perfused while the infant is being rewarmed. The baby is weaned from extracorporeal circulation, and the vent and cannulas are removed (Fig 6). This method minimizes both donor myocardial ischemic and recipient circulatory arrest times.

Results

Total donor myocardial ischemic and recipient circulatory arrest times for infant orthotopic cardiac transplantation in patients with type I hypoplastic left heart syndrome are shown in Table 1. In addition, the length of time from arrival of the donor heart at the hospital to removal of the cross-clamp (hospital ischemic time) is shown.

Comment

The description of infant orthotopic cardiac transplantation for hypoplastic left heart syndrome by Bailey and associates [3] is based on pulmonary artery cannulation and single right atrial venous cannulation, deep hypothermia and circulatory arrest for the entire implantation

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* Type I refers to infants with hypoplastic left heart, hypoplastic aortic arch, and coarctation [7]. Type II refers to infants with hypoplastic left heart, adequately sized ascending aorta, and no coarctation. An adequately sized ascending aorta is defined by a ratio of 0.8 or greater between the diameter of the ascending aorta just below the innominate takeoff and the diameter of the descending aorta just downstream from the ductus arteriosus.

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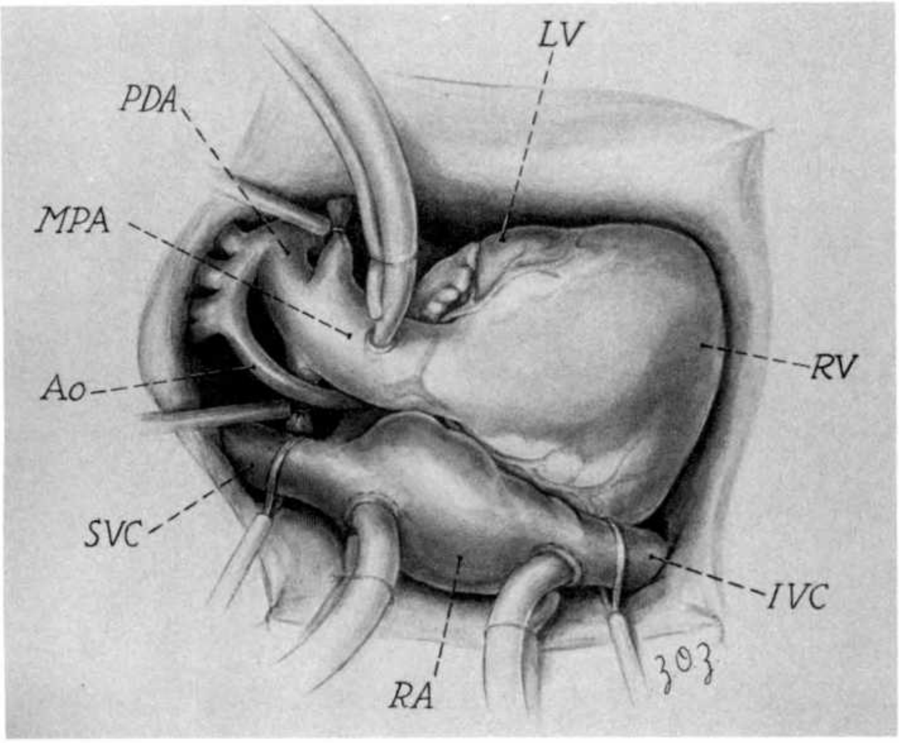


Fig 1. Cannulation for orthotopic cardiac transplantation in an infant with hypoplastic left heart syndrome (type I). Arterial perfusion is via an 8F DLP catheter in the main pulmonary artery (MPA)*. Note snares around right and left pulmonary arteries to prevent flow to the lungs. Venous return is with bicaval cannulation, 12F cannula in the superior vena cava* (SVC) *and 16F cannula in the inferior vena cava* (IVC)*.* (Ao = *aorta;* LV = *left ventricle;* PDA = *patent ductus arteriosus;* RA = *right atrium;* RV = *right ventricle.)*

procedure, and extensive aortic arch reconstruction. This method has yielded excellent short-term results with an average circulatory arrest time of 56 minutes (range, 47 to 65 minutes) [1]. These data notwithstanding, we reasoned that modifications of this technique could decrease total time of donor myocardial ischemia and total time of recipient circulatory arrest.

It is clear from adult registry data [8] that the 30-day mortality increases from 10% for 1 to 2 hours of donor myocardial ischemia to 17% for 4 to 5 hours of donor

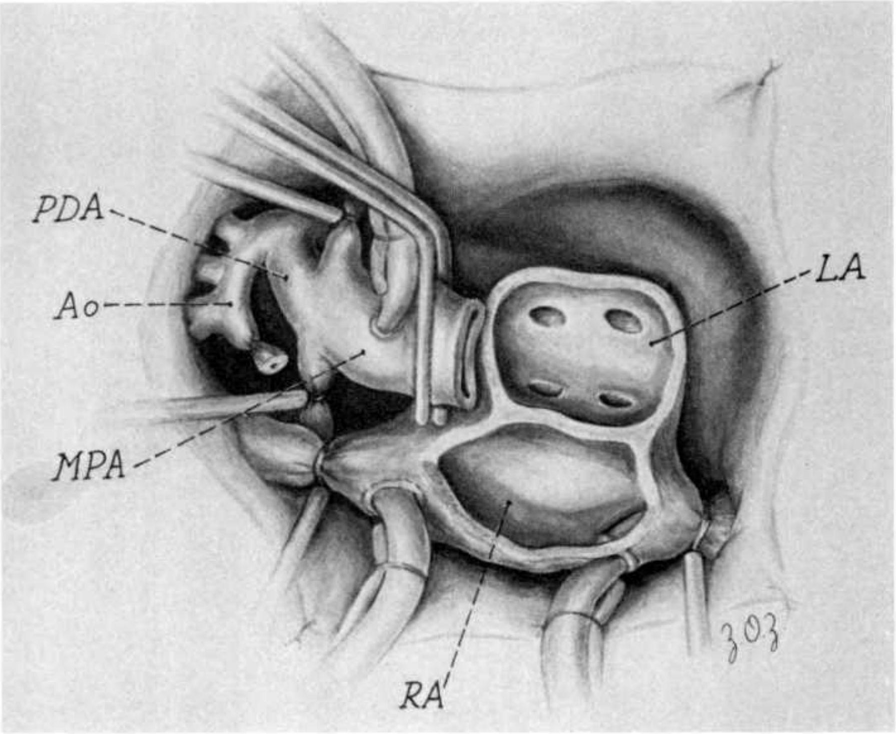
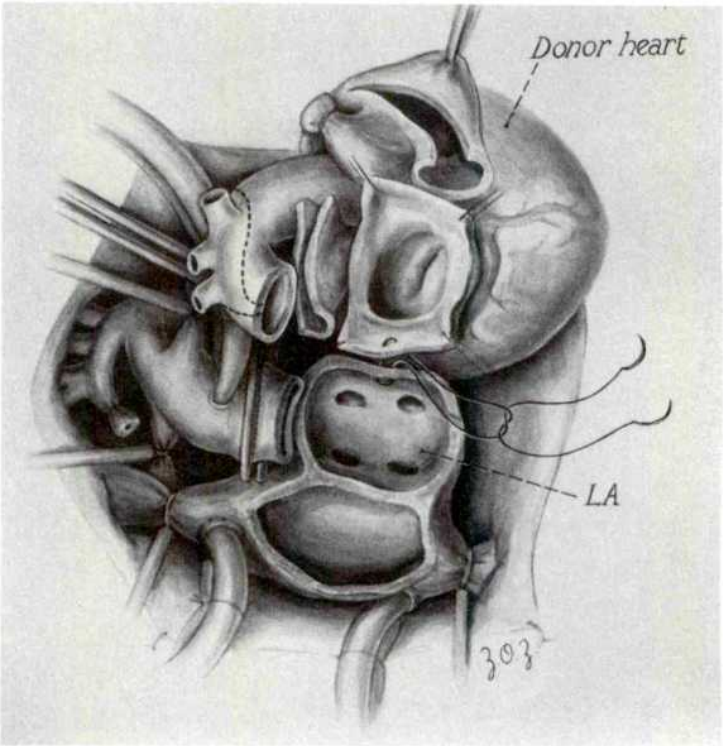


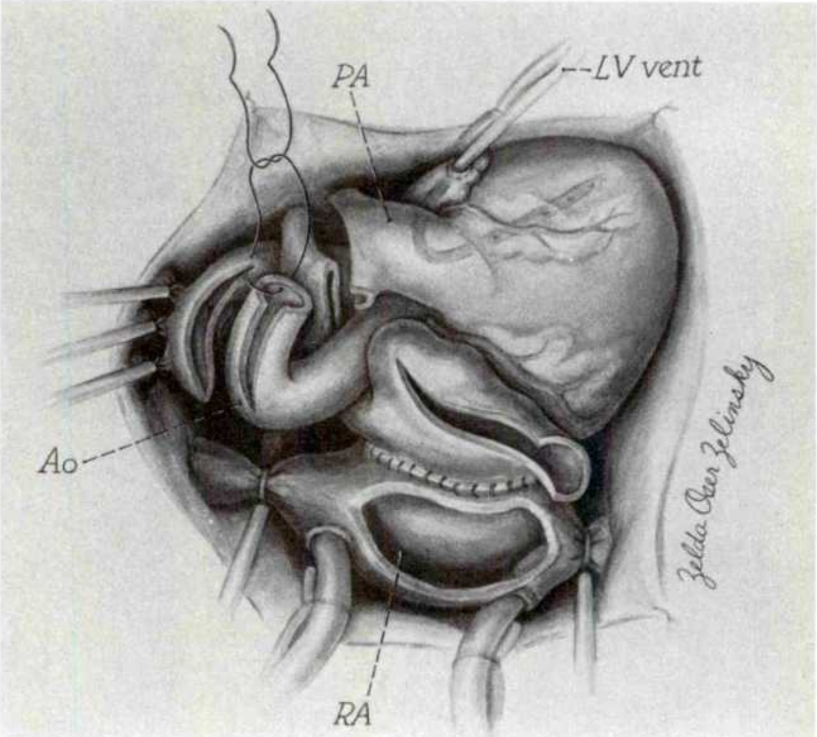
Fig 2. The heart has been excised after placement of a cross-clamp on the main pulmonary artery (MPA) *and ligation and division of the tiny ascending aorta* (Ao)*. Excision of the donor heart is performed with extracorporeal circulation continuing.* (LA = *left atrium;* PDA = *patent ductus arteriosus;* RA = *right atrium.)*

Fig 3. The donor heart is brought into the field, and the left atrial anastomosis is performed while extracorporeal circulation is maintained. The donor arch is tailored as indicated by the dotted lines. *(LA = left atrium.)*



myocardial ischemia. These data may not be directly applicable to infant transplantation, but, in general, we believe that efforts to minimize total time of donor myocardial ischemia should improve results. To this end, we adopted the implantation technique of Cabrol and coworkers [9], which emphasizes an anastomotic sequence change. The left atrial and aortic suture lines are placed first; then aortic cannulation and cardiopulmonary bypass are resumed, and the right atrial and the pulmonary artery anastomoses are completed during the reperfusion

Fig 4. An 8F catheter has been placed in the left ventricle for instillation of cold saline solution. Circulatory arrest is initiated after the head vessels have been controlled by temporary occlusion. The ductus arteriosus is ligated and divided and the main pulmonary artery (PA) transected proximal to the bifurcation. The recipient aortic arch is prepared by aortotomy across the transverse arch and coarctation site (distal to the ligated patent ductus arteriosus). (Ao = aorta; LV = left ventricular; RA = right atrium.)



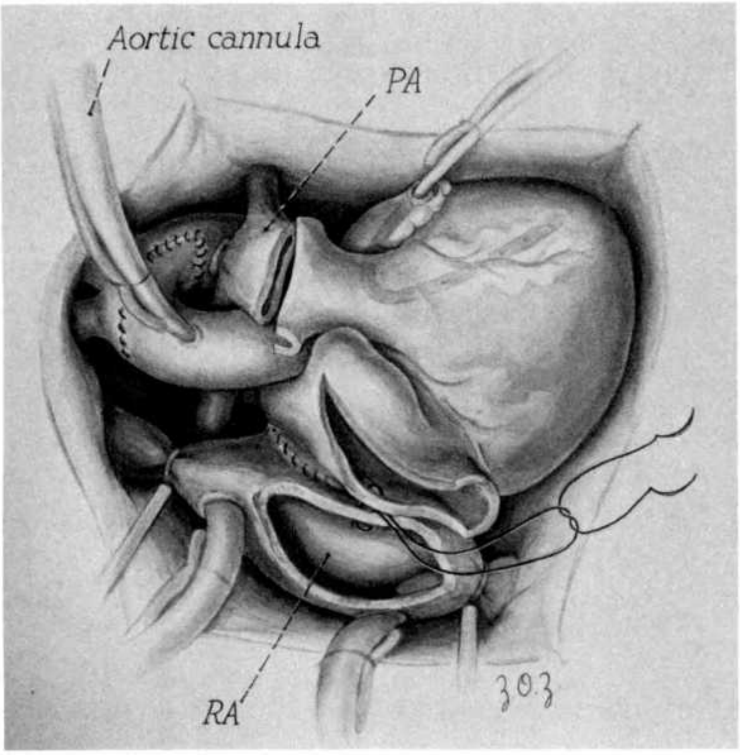


Fig 5. The aortic arch reconstruction is completed, procedures to remove air are performed, and the arch is cannulated (8F DLP), reinstituting cardiopulmonary bypass. The right atrial and the pulmonary artery (PA) *anastomoses are performed with the heart perfused while the infant is rewarmed. The catheter in the left ventricle is now used as a vent.* (RA = *right atrium.)*

and rewarming stage. This technique has resulted in an average 59-minute hospital ischemic time (see Table 1) for our infants with hypoplastic left heart syndrome who required extensive arch reconstruction (type 1).

To minimize the total circulatory arrest time, we use bicaval cannulation and continued extracorporeal circulation for both the excision of the heart and the left atrial anastomosis. Total circulatory arrest is used only for the aortic arch reconstruction. This has produced total circulatory arrest times ranging from 24 to 35 minutes (mean

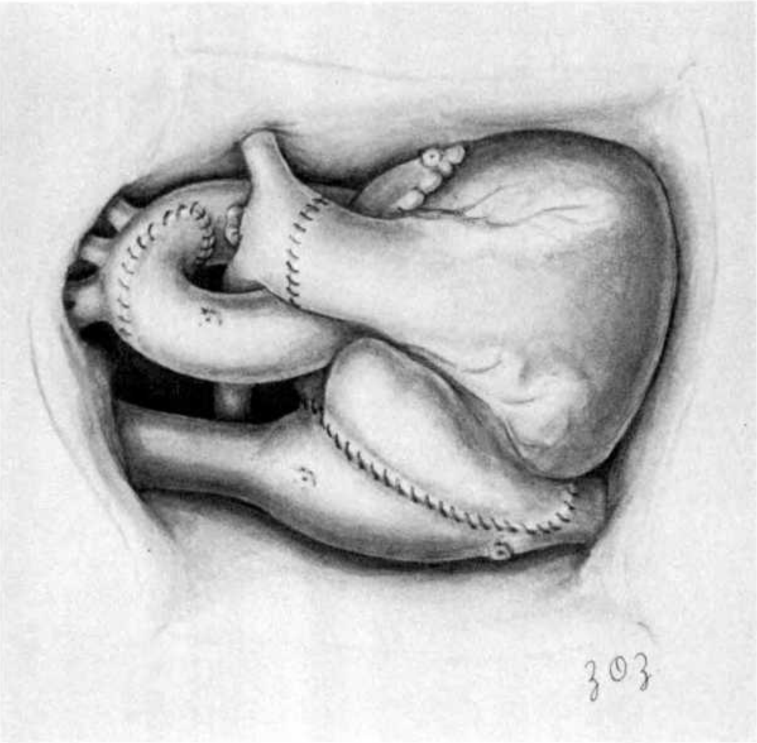


Fig 6. Cardiac transplantation has been completed, and the cannulas have been removed.

Table 1. Myocardial Ischemic and Circulatory Arrest Times for Orthotopic Cardiac Transplantation in Patients With Type I Hypoplastic Left Heart Syndrome

Patient No.	Transport Ischemia (min)	Hospital Ischemia[a] (min)	Donor Ischemia (min)	Circulatory Arrest (min)
1	75	75	150	35
2	108	66	174	32
3	150	48	198	32
4	132	48	180	24
Mean	116	59	176	31

[a] This is the length of time from arrival of the donor heart at the hospital to removal of the cross-clamp near the end of the operation.

duration, 31 minutes), with no operative deaths or recognized neurological deficits. Clinical studies have not clearly determined the "safe" period of circulatory arrest under deep hypothermia for infants undergoing repair of congenital heart disease. However, guidelines have been established to predict a safe period of circulatory arrest of 45 minutes for at least 75% of patients [10]. Some structural evidence of neurological damage can be expected in the majority of patients when the arrest time exceeds 60 minutes [10]. The shorter circulatory arrest times achieved by our method should be well within the safe period for most patients. Furthermore, should technical difficulties arise with the left atrial or the aortic anastomosis, a margin of safety is provided in the event that additional time is needed to resolve those difficulties.

We have described a modified technique of orthotopic cardiac transplantation for hypoplastic left heart syndrome that will allow shorter total donor myocardial ischemic and total recipient ciculatory arrest times. Theoretically, this should allow acceptance of donors from a wider geographical area, improve graft function and recipient survival, and decrease the potential neurological hazards of total circulatory arrest in patients undergoing orthotopic cardiac transplantation for hypoplastic left heart syndrome.

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Tracheal Reconstruction With Polytetrafluoroethylene Graft in Dogs

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Use of prosthetic materials for long-segment tracheal reconstruction has been limited owing to infection, graft migration, ingrowth of fibrous tissue, and stenosis. Polytetrafluoroethylene (PTFE) is flexible and porous, and it may resist infection more than previously used materials. We evaluated PTFE for use in long-segment tracheal reconstruction. A 5-cm segment of trachea was resected in 9 dogs and replaced with a 20-mm reinforced PTFE graft using 4-0 Vicryl sutures. In 2 control dogs, one tracheal arch was resected and a primary anastomosis was performed. The animals were followed up with weekly bronchoscopy and endoscopic photography. Euthanasia was performed at 16 weeks or when signs of respiratory distress developed. At postmorten examination, the anastomoses were examined grossly and with light and scanning electron microscopy. In all 9 dogs that underwent tracheal replacement with PTFE, granulation tissue developed at the anastomoses resulting in airway obstruction after 3 to 8 weeks. No epithelial growth occurred over the graft between the anastomoses. The control animals did well. We conclude that granulation tissue formation at the anastomosis and the lack of respiratory epithelial ingrowth across the graft makes PTFE unsuitable for long-segment tracheal reconstruction.

(Ann Thorac Surg 1990;50:899–901)

Primary anastomosis of the trachea is the procedure of choice for tracheal reconstruction. Successful primary tracheal reconstruction is dependent on adequate blood supply to the tracheal margins and a tension-free anastomosis. This may be difficult to achieve with more extensive tracheal resections or injuries, and attempts at primary repair may result in stenosis secondary to excessive granulation and scarring [1]. Numerous prosthetic materials have been used for tracheal reconstruction but have met with limited success owing to infection, graft migration, or eventual stenosis [2–4].

Polytetrafluoroethylene (PTFE) is commonly used in vascular surgery. Tube grafts made from PTFE are flexible, and the porosity of the material allows for tissue ingrowth. Reinforcing rings outside the prosthesis, similar to those of tracheal arches, help keep the tube widely patent. Polytetrafluoroethylene has also been shown to be more resistant to infection than some other materials [5]. The use of PTFE for tracheal reconstruction has not been investigated fully, and the data available are contradictory [6–8]. The present study was performed to evaluate PTFE for long-segment tracheal reconstruction.

Material and Methods

Eleven mongrel dogs weighing 16 to 31 kg were used. All animals received humane care in compliance with the "Principles of Laboratory Animal Care" formulated by the National Society of Medical Research and the "Guide for the Care and Use of Laboratory Animals" prepared by the National Institutes of Health (NIH publication No. 85-23, revised 1985). The dogs were anesthetized with nitrous oxide, oxygen, and halothane. Electrocardiogram, blood pressure, and serial arterial blood gases were monitored throughout the procedure.

The intrathoracic trachea was exposed through a right thoracotomy and the trachea was mobilized. In 9 dogs, the trachea was transected 2 to 3 cm proximal to the carina, and the left main bronchus was intubated through the surgical field. A 5-cm segment of trachea (7 to 8 tracheal arches) was resected.

The trachea was reconstructed using a 20-mm reinforced PTFE graft. A proximal end-to-end anastomosis was performed using 4-0 Vicryl (Ethicon, Somerville, NJ) sutures, which were tied on the outside of the anastomosis. The anastomosis was created such that the tracheal edge was invaginated within the graft approximately 5 mm and was tension-free. The distal anastomosis was performed using the same technique. The graft was then covered with pleura. In the 2 control dogs, one tracheal arch was resected and a primary anastomosis was performed using the same technique as in the experimental group.

A thoracostomy tube was placed, connected to a Heimlich valve, and removed 48 hours postoperatively. The

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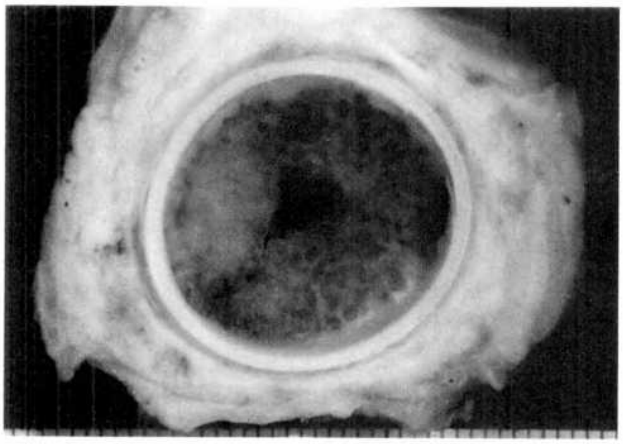

Fig 1. Cross-sectional view through graft shows near-occlusion of the distal anastomosis with granulation tissue at 8 weeks.

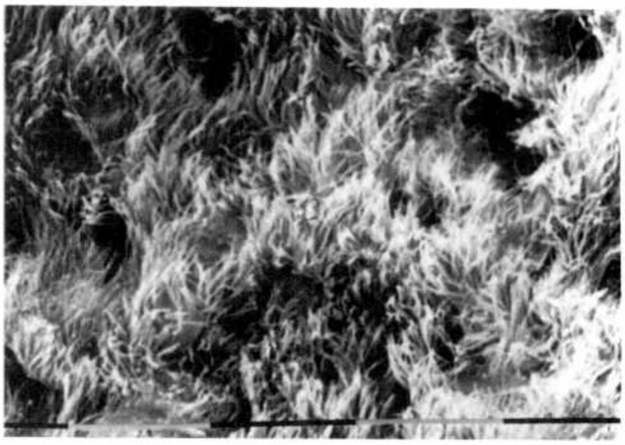

Fig 3. Scanning electron micrograph of normal canine trachea. (×2,720 before 50% reduction.)

animals were given cefoxitin, 15 mg/kg, intravenously at induction of anesthesia and every 8 hours for 3 weeks postoperatively and analgesics as needed.

The animals underwent weekly bronchoscopy with photographic documentation of the trachea. The animals were killed at 16 weeks postoperatively, or earlier if they showed signs of respiratory distress. At postmortem examination, the grafts were resected and examined grossly and with light and scanning electron microscopy.

Results

Bronchoscopic examination in the immediate postoperative period showed the anastomoses to be widely patent. In the experimental animals a smooth glistening layer developed that covered most of the graft after 1 week. In all dogs that underwent tracheal replacement with PTFE granulation tissue developed at both the proximal and distal anastomosis. This granulation tissue resulted in substantial airway obstruction between the third and eighth week, which required euthanization of the dogs (Fig 1). Most of the dogs had copious mucopurulent secretions and pneumonia developed in 5 dogs. None of the dogs had subcutaneous emphysema, air leak, or erosion of the graft into surrounding structures.

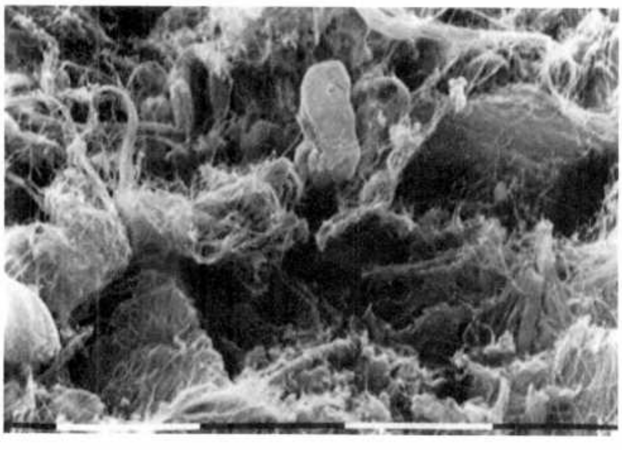

Fig 2. Scanning electron micrograph through mid-graft 8 weeks after implantation. The graft is lined with fibrin and bacteria. (×2,720 before 50% reduction.)

Gross examination of the prostheses at postmortem examination showed a dense fibrous reaction surrounding the graft. The grafts were lined with a smooth, glistening, gray amorphous layer. Light and electron microscopy showed this layer to be comprised of fibrin, inflammatory cells, and bacteria (Fig 2). There was no evidence of respiratory epithelial growth beyond the anastomosis.

No granulation or stenosis developed at the anastomosis in the control animals, and the anastomosis remained widely patent at 16 weeks. Electron microscopy near the anastomosis in the control animals showed normal respiratory epithelium (Fig 3).

Comment

Primary tracheal anastomosis may not be feasible in cases of extensive tracheal resection for tumors, trauma, tracheomalacia, or stenosis, or if extensive scarring from mediastinitis or irradiation prevents mobilization of the trachea. Numerous prosthetic materials have been used experimentally [9–11] and clinically [4, 12, 13] in an attempt to bridge long tracheal defects. Prosthetic tracheal substitution has been generally unsuccessful because the foreign material becomes infected and granulation tissue forms and obstructs the airway. Additionally, several prosthetic materials are quite rigid and can erode into blood vessels resulting in exsanguinating hemorrhage [2–4].

The ideal tracheal prosthesis should be flexible to prevent erosion into major blood vessels, allow for respiratory epithelial ingrowth, be resistant to bacterial invasion, and have minimal tissue reaction. Polytetrafluoroethylene is flexible, and surrounding rings of fluorinated ethylene propylene make the graft noncollapsible. As a vascular prosthesis, the graft is resistant to infection and is associated with minimal tissue reaction.

The use of PTFE for tracheal replacement has been examined in three previous studies. Bottema and Wildevuur [8] replaced 1-cm segments of trachea in rabbits with nonreinforced PTFE. Although graft infection was common and granulation tissue formed at the center of the graft, epithelial growth across the graft was observed after 2 to 4 weeks. This epithelial ingrowth was attributed to the anastomotic technique, which invaginated the tracheal mucosa within the graft.

Recently, Shaha and associates [6] replaced cervical tracheal segments of 7 to 8 arches with reinforced PTFE graft. They noted no mucosal lining of the graft and concluded incorporation of PTFE graft does not occur. However, unlike in the study of Bottema and Wildevuur, the trachea was anastomosed outside the graft. Because no epithelial growth occurred with the trachea outside the graft, we believed that tracheal invagination would be important for epithelialization.

Trojan and co-workers [7] replaced 5-cm segments of trachea with reinforced PTFE graft using an anastomotic technique similar to ours. They reported long-term survival in several animals and epithelialization of the graft. As in our study, the graft was lined with bacteria and fibrin for the first 2 weeks; however, this was replaced with a continuous layer of flat epithelium between 2 and 4 weeks, and pseudorespiratory epithelium between the fifth and sixth week. An endoscopic photograph of the distal anastomosis at 4 weeks in the report by Trojan and co-workers shows a stenosis identical to that seen in our experimental animals. Because the anastomotic technique and early results between Trojan's study and our study are identical, it is unclear why they observed a cessation of the stenosis and epithelialization of the graft whereas we observed progression of the stenosis and no epithelialization.

Although steroids and cauterization of granulation tissue at tracheal anastomoses have been used clinically to treat stenoses, we were interested in the normal healing response at the graft–tracheal interface and did not want to introduce additional variables that would alter this response. We therefore made no attempt to remove or treat the stenoses in our study.

In conclusion, a suture technique that invaginated the trachea within the graft resulted in the development of granulation tissue at the anastomosis and eventual airway obstruction. No epithelial lining developed within the graft. This study indicates PTFE is probably an unsuitable material for long-segment tracheal reconstruction.

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Endothelial Cell Toxicity of Solid-Organ Preservation Solutions

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Endothelial cell damage caused by myocardial cardioplegic solutions (Bretschneider HTK and St. Thomas' Hospital No. 2) or renal and hepatic cold storage solutions (modified Collins and University of Wisconsin solution) was assessed in monolayer cultures of adult human venous endothelial cells at 4° to 10°C with phase-contrast microscopy. St. Thomas' Hospital solution caused the cells to contract, resulting in disruption of monolayer integrity and opening of intercellular gaps, and resulted in a 24-hour postexposure survival of 51.0% ± 2.4%. Bretschneider HTK solution altered cellular morphology less and produced the best postexposure survival (80.2% ± 2.6%; $p < 0.001$). Although morphology was altered the least with University of Wisconsin solution, postexposure survival with this solution, which was similar to that with modified Collins solution, was superior to that with St. Thomas' ($p < 0.01$) but inferior to that with Bretschneider HTK ($p < 0.05$). The superior protection provided by Bretschneider HTK was due to its additives histidine, tryptophan, and KH-2-oxygluterate ($p < 0.005$), and to its low chloride content ($p < 0.005$). Furthermore, modifying St. Thomas' solution by decreasing its chloride content improved cell survival to 71.2% ± 2.3% ($p < 0.001$). Normothermic (37°C) exposure to Bretschneider HTK, modified Collins, and University of Wisconsin solution was cytotoxic, whereas normothermic exposure to St. Thomas' cardioplegia was not. In conclusion, the preservation solution that is the least harmful to endothelial cells at hypothermia is Bretschneider HTK cardioplegic solution.

(Ann Thorac Surg 1990;50:902–10)

Techniques used for solid-organ preservation primarily involve decreasing basal cellular energy requirements by selectively manipulating specific energy-consuming processes in conjunction with hypothermia. Thus the majority of centers store kidneys and livers at 4° to 10°C and flush these organs intravascularly with "intracellular" electrolyte equivalent preservation solutions to abolish energy-consuming transmembranous electrolyte gradients. Simple hypothermic storage periods of 12 to 24 hours are now clinically achievable for the liver and kidney.

In contrast, the myocardium has a much higher rate of energy consumption because of myocyte electromechanical activity. Thus cardioplegic solutions have been primarily formulated to produce diastolic arrest by increasing the extracellular concentration of potassium and magnesium, or alternatively decreasing the concentration of sodium and calcium. Despite electromechanical arrest the heart is only viable clinically for 4 to 8 hours with simple hypothermic storage. Such periods have been achieved with both intracellular and extracellular electrolyte equivalent cardioplegic solutions.

The endothelium, which is common to all organs, is possibly even more vulnerable to ischemia than cardiomyocytes [1]. Endothelial cells form the primary interface between the blood and interstitium, and modulate a variety of biologic functions [2]. The normal endothelial surface is antithrombogenic and also provides mediators that modulate vascular tone [3]. Ischemia and intravascular infusion of solutions can cause endothelial damage [4–6]. Injury can result in a procoagulant endothelium, vasoconstriction [7, 8], release of enzymes associated with the production of toxic free radicals, increased vascular permeability which predisposes to interstitial edema [9], and accelerated atherogenesis [10]. Furthermore, gradual cell damage leads to the manifestation of these deleterious effects before total loss of the endothelium occurs [11]. Therefore, it is extremely important that organ preservation solutions express minimal toxicity to endothelial cells, and they should in fact protect this structure. The preservation of the endothelium might well be a dominant factor in lung storage, if one considers the extent of the pulmonary vascular bed.

To date, few studies have determined to what extent commonly used preservation solutions influence the endothelium. Furthermore, electrolyte concentrations in cardioplegic solutions are specific for the myocyte and are not necessarily suitable for the preservation of endothelial cells, which do not have inward voltage-dependent sodium or calcium currents [12]. Thus modifications that additionally diminish basic membrane energy requirements, improve buffering, or diminish cellular edema might be of more direct benefit to the endothelium.

This study was undertaken to examine the effects of

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prolonged endothelial cell contact with various standard solutions used for myocardial, renal, and hepatic preservation. Endothelial cells are attachment-dependent cells, and cytotoxicity can be observed in monolayer cultures with light microscopy as loss of cellular attachment and cellular disruption. We elected to study human venous endothelial cells as they were easily obtainable and the majority of endothelial functions stated herein occur universally throughout the vascular tree.

We examined the effect of these solutions both at 4° to 10°C and at 37°C, as the majority of centers infuse these solutions at a temperature of 4°C into the normothermic (37°C) organ to induce hypothermia (4° to 10°C). In addition, modified cardioplegic solutions are also used at normothermia as reperfusion solutions. Finally, in an attempt to establish the primary components that were either harmful or beneficial, modifications to these solutions were developed and tested.

Material and Methods

Endothelial Cell Culture Techniques

Endothelial cells were harvested from human saphenous vein segments obtained from adult patients undergoing coronary artery bypass grafting or from organ transplantation donors as previously described [13]. After cannulation, the venous segment was flushed with incomplete culture medium (Medium 199—Earle's salts; Flow Laboratories, Irvine, Scotland), then filled with 0.1% collagenase (CLS II; Cooper Biomedical, Malvern, PA) and incubated at 37°C for 15 minutes. The collagenase-cell suspension was then inactivated by flushing with culture medium containing 30% fetal calf serum (Delta Bioproducts, Johannesburg, South Africa), collected, and centrifuged for 10 minutes at 120 *g*. The pellet was resuspended in 2 mL of complete culture medium (Medium 199 plus 20% fetal calf serum and endothelial cell growth factor; Collaborative Research Inc, Lexington, MA) and plated into a 9.6-cm^2 well of a six-well culture plate precoated with human fibronectin (Inotech, Zurich, Switzerland; 18 μg/mL). All cultures were incubated at 37°C in a 5% CO_2 atmosphere.

To remove erythrocytes and cell debris the primary culture was rinsed after 24 hours with Dulbecco's phosphate-buffered saline (Flow Laboratories). Thereafter, complete culture medium (2 mL) was added, and 50% of the supernatant was replaced twice weekly with fresh complete culture medium. Endothelial cells were passaged at 80% partial cell coverage, designated by a micro-grid technique [13]. This partial cell coverage was usually obtained 10 days after endothelial cell harvest.

To reduce the cell-damaging effect of multiple passages [14], primary cultures were passaged at a ratio of 1:22, using trypsin ethylenediaminetetraacetic acid (Flow Laboratories). These first-passage endothelial cells were either seeded into a 175-cm^2 flask precoated for 12 hours with gelatin (Difco Laboratories, Detroit, MI; 10 ng/mL in 0.1 mol/L $NaHCO_3$ buffer) or onto 9-mm glass cover-slips for immunohistochemical staining with von Willebrand factor (factor VIII–related antigen), which is a typical cell marker for endothelial cells [15]. Cultures were managed as before, and confluence was again obtained within 10 days.

Table 1. Composition of Preservation Solutions[a]

Component	ST	COL	UW-CSS	B-HTK
Na	120	10	20	15
K	16	115	140	10
HCO_3	10	10	. . .	. . .
Mg	16	3	5	4
Ca	1.2	. . .	. . .	. . .
Cl	159	15	. . .	50
Glucose · H_2O		139		
Histidine HCl · H_2O				18
Histidine				180
Tryptophan				2
KH-2-oxygluterate				1
Mannitol				30
Lactobionic acid			100	
Raffinose · $5H_2O$			30	
Adenosine			5	
Glutathione (reduced)			3	
Allopurinol			1	
Pentafraction			50 g/L	
Osmolarity (mosmol/L)	300	300	320	298
pH (37°C)[b]	7.26	7.27	7.27	7.33

[a] Concentrations are given in mmol/L, unless otherwise indicated.
[b] The pH was measured after gassing with 95% O_2 and 5% CO_2.

B-HTK = Bretschneider HTK; COL = modified Collins solution; ST = St. Thomas' Hospital No. 2; UW-CSS = University of Wisconsin cold storage solution.

The cells were passaged once more, but in a 1:1 ratio into 12-well culture plates (3.5 cm^2 per well) precoated with gelatin. Confluence of second-passage monolayers was obtained after 2 to 3 days. Individual wells were assessed microscopically for completeness and homogeneity of cell coverage, and unevenly covered wells were excluded.

Preservation Solutions

Preservation solutions tested were the St. Thomas' Hospital No. 2 (ST; Sabax Ltd, Johannesburg, South Africa) and Bretschneider HTK (B-HTK; Dr F Koehler Chemie GmbH, Alsbach, Federal Republic of Germany) cardioplegic solutions, the modified Collins (COL) kidney preservation solution, made up by adding 50 mL of 50% dextrose and 1.2 mL of 50% $MgSO_4$ to the standard Collins solution (Baxter Health Care Corporation, Deerfield, IL), and the University of Wisconsin cold storage solution (UW-CSS; Du Pont Pharmaceuticals, Wilmington, DE), but without the recommended additional additives (penicillin, insulin, dexamethasone). The compositions of these solutions are given in Table 1.

Modifications of these primary preservation solutions

Table 2. Composition of Modified Experimental Solutions[a]

Component	ST	ST-Low Cl	ST-Ca (?)	Incell High Cl	No HTK
Na	120	120	120	10	15
K	16	16	16	126	10
HCO_3	10	10	10	10	. . .
Mg	16	16	16	16	4
Cl	159	34	158	158	50
Ca	1.2	1.2	0 0.05 0.6	. . .	. . .
Gluconate		126			
Mannitol					30
Sucrose					208
Osmolarity (mosmol/L)	300	276	300	288	312
pH (37°C)[b]	7.26	7.28	7.23	7.27	7.27

[a] Concentrations are given in mmol/L, unless otherwise indicated.
[b] The pH was measured after gassing with 95% O_2 and 5% CO_2.

Incell-High Cl = an experimental intracellular electrolyte equivalent solution with a high chloride ion content. No-HTK = Bretschneider HTK cardioplegic solution, but without histidine, tryptophan, and KH-2-oxygluterate. Sucrose was used to maintain equivalent osmolarity. ST = St. Thomas' Hospital No. 2 cardioplegic solution, and modifications as indicated.

were made up in our laboratory to examine the specific effect of chloride ion concentration and to examine B-HTK in more detail. St. Thomas' cardioplegia with a low chloride concentration (ST-Low Cl) was made with sodium and potassium gluconate instead of the respective chloride salt. However, gluconate binds calcium and therefore affects the free ionized calcium ion content, thus ST with varying calcium concentrations (ST-Ca 0 mmol/L, 0.05 mmol/L, 0.6 mmol/L) were also tested. The "free" ionized calcium of these solutions was measured using a calcium-ion–selective electrode (Ciba Corning 288 Blood Gas System, Ciba Corning Diagnostics Corporation, Medfield, MA). However, because of the high magnesium concentration of ST, an estimate of the magnesium ion interference in the calcium ion measurements had to be subtracted from the initial values. The estimate was obtained by measuring the "ionized calcium" in two acalcemic solutions that did or did not contain magnesium (16 mmol/L) but were otherwise identical to the corresponding modified ST solution. This "corrected" estimate of the free ionized calcium is the reported value. In addition, an acalcemic "intracellular" solution with a high chloride content (Incell-High Cl) was prepared. Apart from a substantially higher chloride concentration this solution was not markedly dissimilar to COL (Tables 1, 2). Finally, a solution matching the electrolyte content of B-HTK but without the additives histidine, tryptophan, or KH-2-oxygluterate (No-HTK) was prepared. Sucrose was used in No-HTK to maintain equivalent osmolarity. The compositions of these experimental solutions are given in Table 2.

All solutions were gassed with 95% O_2 and 5% CO_2 30 minutes before use to ensure pH stability and uniform oxygen content. Gassing with 95% O_2 and 5% CO_2 will induce a slight acidotic pH shift if the primary buffer in the solution is bicarbonate (ST, COL). The pH of the ST cardioplegic solution was therefore slightly acidotic (pH, 7.26 at 37°C) compared with the value reported for clinical use, but was similar to that of the other solutions (Tables 1, 2). However, we have previously shown that this acidotic shift in pH improves postischemic recovery of the isolated rat heart after protection with an oxygenated ST plus glucose (11 mmol/L) cardioplegic solution [16].

Experimental Protocol

A baseline in situ grid count of each well was performed [13], using an inverted-phase contrast microscope (ID 02 MT, Zeiss, Oberkochen Federal Republic of Germany). The cell count in "20 grid squares" was the total number of cells seen in each of the four corner squares of a marker grid and counted in five different areas of each well. The culture medium was then removed and the preservation solution applied initially as a rinse to ensure removal of all medium, and then a 1-ml aliquot was reinserted.

In the normothermic experiments, preservation solutions at room temperature were introduced onto the cells, which were then incubated at 37°C in a 5% CO_2 atmosphere. Preservation solutions for the hypothermic experiments were cooled to 10°C before application, and the cultures were then stored in a refrigerator at 4° to 10°C in a normal atmosphere.

The cells were exposed to the preservation solutions for 12 hours, and the morphology of the cells was then examined by light microscopy. Thereafter the solution was removed and the cells were rinsed with incomplete culture medium; and 1 mL of complete medium, but without endothelial cell growth factor, was reapplied. All cell cultures were again placed in a 37°C, 5% CO_2 incubator to assess cell survival and allow for any delayed expression of cell damage. A postexposure cell count was performed after the cells were rinsed with incomplete culture medium, to remove debris and dead cells, at 24 hours and again at 36 hours.

Expression of Results

The cell count per square centimeter was calculated at each of three time periods from in situ microgrid counts as previously described [13]. The percentage of surviving cells in each well at the 24-hour and 36-hour postexposure time periods was derived from the ratio of each individual postexposure count to the corresponding initial preexposure count for that well. Values are presented as survival percentage means and standard errors of survival percentage means.

The total number of wells tested is given as N, and to ensure reproducibility each solution was tested in a number of different culture plates (P). Cells were obtained from three separate human donors, and we attempted to test each solution on cells from at least two different primary cultures (C), where these were available. Student's t test was used to compare differences on the

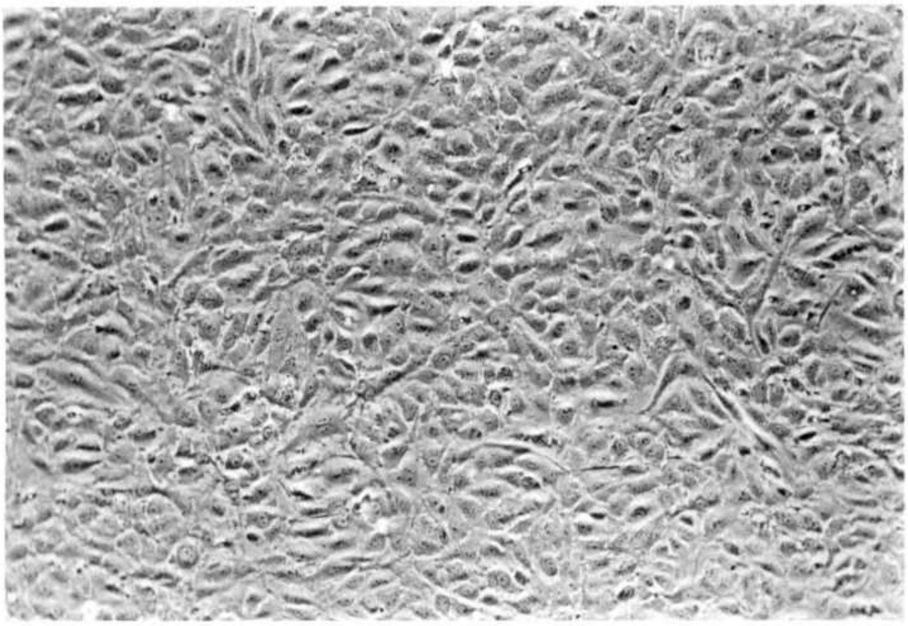

Fig 1. Phase contrast microscopic appearance of a confluent monolayer of adult human saphenous vein endothelial cells. Typical cobblestone morphology and monolayer growth of a pure endothelial cell culture are shown. (×100 before 25% reduction.)

assumption of equal variances. Appropriate tables were then used to determine p values. Statistical significance was assumed when the p value was less than 0.05. Several comparisons were of a priori interest, and the stated level of significance was used without multiple comparison adjustments on the basis of the a priori intentions. Many of the p values of interest are exceptionally small, and even the crudest adjustment for multiple comparisons will leave the conclusion of significant differences unchanged. The wells were treated as though they demonstrably generated independent observations. Although this is not theoretically optimal, the low variation between primary cultures in the same treatment regimen ensures that the approximate method generates valid conclusions for these data.

Results

The phase contrast microscopy appearance of a confluent monolayer of cultured human endothelial cells is shown in Figure 1, which shows the typical "cobblestone" morphology.

Hypothermic Exposure (10°C)

Exposure at 10°C for 12 hours to ST (N = 16, P = 5, C = 3) caused the cells to contract, and denser cytoplasmic granulations and intercellular gaps were seen (Fig 2). However, this appearance was reversible after removal of the cardioplegic solution and reincubation in culture medium, and the 24-hour postexposure survival was 51.0% ± 2.4%. The 36-hour survival was no different (Table 3). In contrast, exposure to B-HTK (N = 13, P = 4, C = 3) altered the morphological appearance of the endothelial cells less; contraction of the cells was less but intercellular gaps still appeared. The 24-hour postexposure endothelial cell survival was 80.2% ± 2.6%; after 36 hours it was slightly lower at 73.5% ± 1.6% ($p < 0.05$). This was superior to ST ($p < 0.001$).

Exposure to UW-CSS did not alter the cells' appearance and preserved their morphology the best, whereas COL induced prominent nuclei with otherwise minimal cellular changes. Collins solution (N = 9, P = 3, C = 2) was associated with a 24-hour endothelial cell survival of 62.4% ± 3.1%, which was no different than the 69.7% ± 2.9% survival of cells exposed to UW-CSS (N = 13, P = 4, C = 3). Cell survival with both these alternative solid-organ preservation solutions was better than with ST ($p < 0.01$), but inferior to that with B-HTK ($p < 0.05$ at both postexposure periods) (see Table 3).

MODIFICATIONS TO BRETSCHNEIDER HTK CARDIOPLEGIC SOLUTION. When endothelial cells were exposed for 12 hours at 10°C to No-HTK (see Table 1), the 24-hour cell survival was 69.0% ± 2.3% (N = 9, P = 3, C = 2). This survival was inferior to that with the standard B-HTK formulation ($p < 0.01$) and no different than that with COL (see Table 3). The morphological appearance of the cells was similar to those treated with B-HTK.

MODIFICATIONS TO INTRACELLULAR PRESERVATION SOLUTIONS. Incell-High Cl (N = 11, P = 3, C = 2) is an intracellular electrolyte equivalent solution with a high chloride concentration, and produced a diminished 24-hour endothelial cell survival of 43.6% ± 4.5% ($p < 0.005$), in contrast to COL (see Table 3). This high concentration of chloride caused the cells to contract, and intercellular gaps were now easily seen, similar to the morphology observed with ST.

MODIFICATIONS TO ST. THOMAS' CARDIOPLEGIC SOLUTION. Endothelial cells were exposed for 12 hours at 10°C to ST-Low Cl (N = 11, P = 4, C = 2) and 24-hour survival increased to 71.2% ± 2.3% ($p < 0.001$ compared with ST, which has a high chloride content [see Tables 2, 3]), but ST-Low Cl was still inferior to B-HTK ($p < 0.05$). Although the postexposure morphological appearance of cells ex-

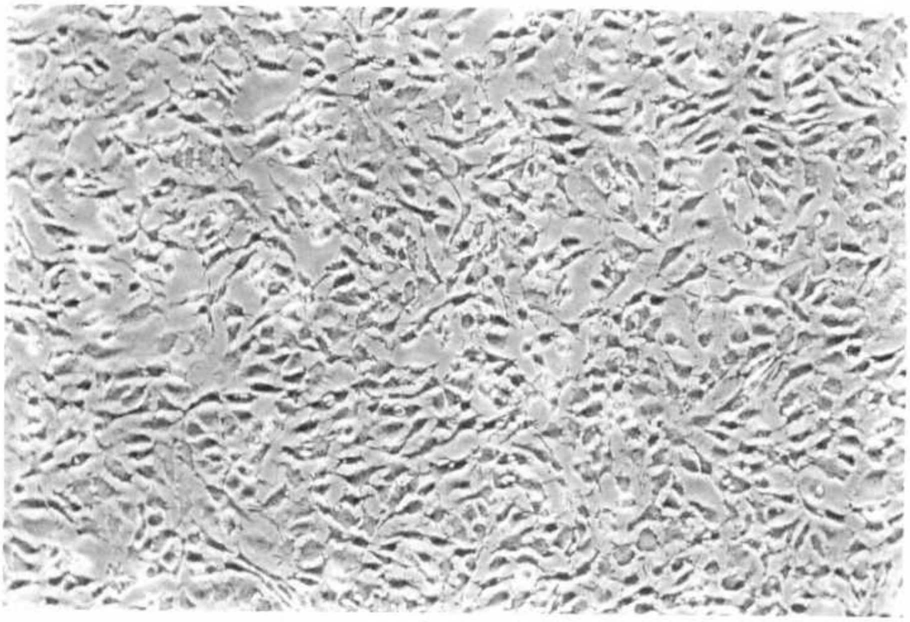

Fig 2. Phase contrast microscopic appearance of a monolayer of adult human endothelial cells after 12-hour exposure at 4° to 10°C to St. Thomas' Hospital No. 2 cardioplegic solution. Endothelial cells show condensed cytoplasmic granulations and have contracted, causing disruption of monolayer integrity and the opening of wide intercellular gaps. (×100 before 25% reduction.)

posed to ST-Low Cl showed some cellular contraction with intercellular gaps, this was less than that seen after exposure to ST.

The anion gluconate (used to replace chloride ions in ST-Low Cl) binds calcium, and the estimated free ionized calcium of ST (0.9 mmol/L) decreased to 0.16 mmol/L in ST-Low Cl. Therefore, to independently examine the effect of a lowered Ca^{2+} concentration in ST, we tested a hypocalcemic ST-Ca 0.6 mmol/L (N = 6, P = 2, C = 2) having a free Ca^{2+} of 0.5 mmol/L, and ST-Ca 0.05 mmol/L solution (N = 7, P = 2, C = 2) with a free Ca^{2+} of less than 0.05 mmol/L. Cell survivals of 60.4% ± 4.2% and 59.9% ± 4.3%, respectively, were obtained 24 hours after exposure, and these hypocalcemic ST solutions were not statistically superior to ST (Fig 3), in contrast to ST-Low Cl. The estimated free ionized calcium of ST-Low Cl (0.16 mmol/L) fell between those of the hypocalcemic ST solutions.

However, an "acalcemic" ST solution, ST-Ca 0 mmol/L (N = 8, P = 3, C = 2) improved 24-hour cell survival to 64.8% ± 2.1% ($p < 0.001$) and 36-hour cell survival ($p < 0.001$) compared with ST. These cells also showed less contraction with smaller intercellular gaps; however, cellular contraction was least with ST-Low Cl. A note of

Table 3. Endothelial Cell Survival After 12-Hour Hypothermic Exposure[a]

Solution	N	Baseline Count (cells/cm²)	% Survival at 24 h	% Survival at 36 h
Primary preservation solutions				
B-HTK	13	21,231 ± 1,780	80.2% ± 2.6%[b]	73.5% ± 1.6%[b]
UW-CSS	13	21,510 ± 2,328	69.7% ± 2.9%[b,c]	65.5% ± 1.5%[b,d]
COL	9	26,486 ± 2,139	62.4% ± 3.1%[d,e]	61.1% ± 2.3%[d,e]
ST	16	20,758 ± 1,545	51.0% ± 2.4%	49.6% ± 2.5%
Modifications				
ST-Low Cl	11	25,557 ± 2,341	71.2% ± 2.3%[b,c]	67.0% ± 2.2%[b,c]
Incell-High Cl	11	26,330 ± 2,423	43.6% ± 4.5%	41.3% ± 4.4%
No-HTK	9	26,306 ± 2,418	69.0% ± 2.3%[b,d]	66.3% ± 1.6%[b,d]

[a] Cultured human endothelial cell survival after exposure to organ preservation solutions for 12 hours at hypothermia (4° to 10°C). The cell survival at 24 h and 36 hours after exposure is presented as a percentage of each individual baseline preexposure cell count per square centimeter. Results are presented as survival percentage means and standard errors of percentage means. [b] $p < 0.001$ compared with ST at corresponding time periods. [c] $p < 0.05$ compared with B-HTK at corresponding time periods. [d] $p < 0.005$ compared with B-HTK at corresponding time periods. [e] $p < 0.01$ compared with ST at corresponding time periods.

B-HTK = Bretschneider HTK cardioplegic solution; COL = modified Collins solution; Incell-High Cl = an experimental intracellular electrolyte equivalent solution with a high chloride ion content; No-HTK = B-HTK without histidine, tryptophan, and KH-2-oxygluterate; ST = St. Thomas' Hospital No. 2; UW-CSS = University of Wisconsin cold storage solution.

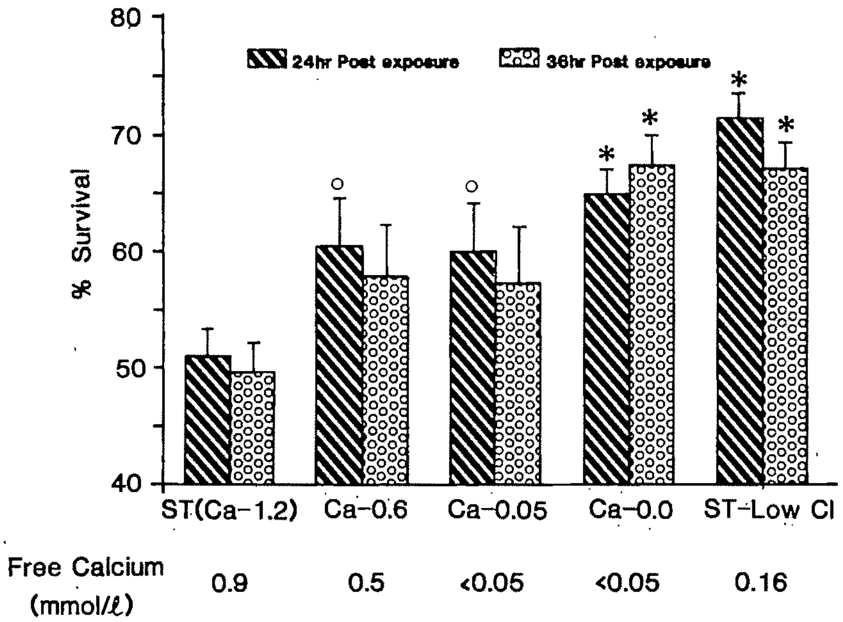

Fig 3. Mean percentage survival of cultured adult human endothelial cells 24 and 36 hours after 12-hour hypothermic (4° to 10°C) exposure to St. Thomas' Hospital No. 2 cardioplegic solution (ST). The effect of varying calcium (Ca) concentrations (0, 0.05, and 0.6 mmol/L) compared with the standard ST (Ca = 1.2 mmol/L) and with an ST solution with a low chloride content (also containing Ca = 1.2 mmol/L) is shown. The vertical bars *represent standard errors of percentage means. The estimated free ionized calcium of each solution is indicated separately. (*p < 0.001 compared with ST(Ca-1.2); °p < 0.05 compared with ST-Low Cl.)*

caution must be mentioned though, as the measured free ionized calcium of ST-Ca 0 mmol/L was not zero; even deionized distilled water contains trace amounts of calcium.

Normothermic Exposure (37°C)

For the normothermic experiments the endothelial cells were maintained for 12 hours at 37°C in a buffered 5% CO_2 atmosphere. In contrast to the hypothermic experiments, ST (N = 9, P = 3, C = 3) was now associated with the best 24-hour endothelial survival of 72.0% ± 2.3% ($p < 0.001$ for all comparisons) (Table 4). In addition, these cells showed only prominent nuclei with otherwise minimal changes in the immediate postexposure period, in contrast to the marked changes observed with hypothermic exposure.

The intracellular preservation solutions, ie, COL (N = 6, P = 2, C = 1), UW-CSS (N = 6, P = 2, C = 2), and B-HTK (N = 9, P = 3, C = 3), all produced immediate loss of adherence and cellular disruption (Fig 4), and 24-hour postexposure endothelial cell survivals were less than 20% (see Table 4). However, the high-chloride intracellular solution Incell-High Cl (N = 6, P = 1, C = 1) was associated with a 24-hour survival of 51.1% ± 3.9% ($p < 0.001$ compared with COL, UW-CSS, B-HTK) (see Table 4).

Table 4. Endothelial Cell Survival After 12-Hour Normothermic Exposure[a]

Solution	N	Baseline Count (cells/cm²)	% Survival at 24 h	% Survival at 36 h
Primary preservation solutions				
B-HTK	9	25,472 ± 3,762	19.1% ± 3.5%[b,c]	18.3% ± 3.4%[b,c]
UW-CSS	6	17,146 ± 825	7.7% ± 1.3%[b,c]	6.7% ± 1.3%[b,c]
COL	6	26,771 ± 952	6.1% ± 0.4%[b,c]	2.8% ± 0.4%[b,c]
ST	9	25,097 ± 3,612	72.0% ± 2.3%	70.1% ± 2.1%
Modifications				
ST-Low Cl	8	27,313 ± 2,410	74.6% ± 1.6%[d]	64.3% ± 1.8%[c]
Incell-High Cl	6	29,333 ± 696	51.1% ± 3.9%[b]	45.8% ± 3.3%[b]
ST-Acalcemic	8	28,828 ± 2,332	66.4% ± 2.0%[d]	56.1% ± 2.1%[b,d]

[a] Cultured human endothelial cell survival after exposure to organ preservation solutions for 12 hours at normothermia (37°C). The cell survival at 24 and 36 hours after exposure is presented as a percentage of each individual baseline preexposure cell count per square centimenter. Results are presented as survival percentage means and standard errors of percentage means. [b] $p < 0.001$ compared with ST at corresponding time periods. [c] $p < 0.001$ compared with Incell-High Cl at corresponding time periods. [d] $p < 0.05$ compared with Incell-High Cl at corresponding time periods.

Abbreviations as in Table 3.

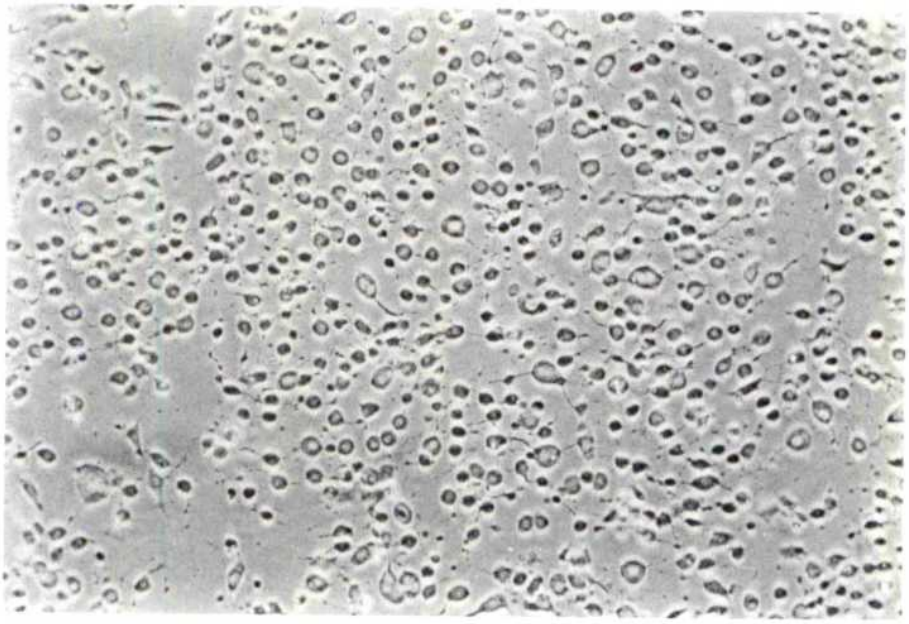

Fig 4. Phase contrast microscopic appearance of a monolayer of adult human endothelial cells after 12-hour exposure at 37°C to modified Collins preservation solution. All endothelial cells are detached from the surface of the culture plate, and the majority show swelling, membrane disruption, or karyorrhexis. (×100 before 27% reduction.)

Finally, exposure to ST-Low Cl ($N = 8$, $P = 3$, $C = 2$) for 12 hours at normothermia produced a postexposure survival of 74.6% ± 1.6%, which was similar to that seen with ST. In addition, the 24-hour survival with ST-Ca 0 mmol/L ($N = 8$, $P = 3$, $C = 2$) of 66.4% ± 2.0% was also no different than that with ST, although at 36 hours it was inferior ($p < 0.001$) (see Table 4). Postexposure morphology was similar to that seen with ST.

Comment

We studied the effect of various internationally used organ preservation solutions on cultured adult human venous endothelial cells. We recognize that arterial endothelial cells differ from venous endothelial cells; however, similarities also exist [17]. Furthermore, many reported differences between cells derived from distinct vascular beds have been observed in various culture conditions and animal models, which in turn may be associated with interspecies differences [18]. We based our model on the assessment of morphological alterations and evaluation of cell detachment, as indicators of cell damage and ultimately cell death. Both these criteria are common to arterial and venous endothelium. Therefore, we believe extrapolation to the arterial system is justifiable.

When confluent monolayers of endothelial cells were exposed to these solutions for 12 hours at hypothermia (4° to 10°C), Bretschneider HTK cardioplegic solution was associated with the best 24-hour postexposure cell survival (80%), although cellular morphology was initially altered by the solution. Bretschneider HTK provided better endothelial preservation than St. Thomas' Hospital No. 2 cardioplegic solution (51%). In addition, both the modified Collins kidney preservation solution and the University of Wisconsin liver and pancreatic storage solution, although superior to ST, were inferior to B-HTK.

Hence the intracellular electrolyte equivalent solutions (B-HTK, COL, UW-CSS) designed to abolish transmembranous electrolyte gradients provided better hypothermic endothelial protection than an extracellular solution (ST). However, the solution that preserved morphology the best (UW-CSS) was not associated with the best postexposure survival.

Nevertheless, if the cells were exposed to these solutions for 12 hours at normothermia (37°C), the intracellular electrolyte equivalent preservation solutions were cytotoxic, with less than 20% 24-hour postexposure survival. St. Thomas' cardioplegia solution was now superior, producing 72% endothelial cell survival.

An atmosphere of 5% CO_2 was used for the normothermic experiments in order to ensure pH stability, because of the substantially lower solubility coefficient of CO_2 at this higher temperature. In our opinion this would have improved normothermic cell survival, and therefore might have accounted for the apparent superior endothelial protection afforded by ST at 37°C, in contrast to 10°C. However, we infer that the observed diminished survival responses with the intracellular solutions at normothermia were determined by the interaction of solution composition and temperature. These contrasting findings possibly support the hypothesis of Kohno and associates [19], who suggest that two types of solutions are required for optimal myocardial preservation, a solution for normothermic arrest and then another for hypothermic preservation. In addition, it would imply that standard cardioplegic solutions (eg, B-HTK) should not necessarily be used as the convenient basic composition for normothermic reperfusion solutions.

To further define the differences between these two groups of solutions we examined various modifications. Primary disparities between intracellular electrolyte equivalent solutions and extracellular solutions are acalcemia, low sodium concentration, and low chloride concentration of the former.

Calcium and Sodium

A sodium-poor solution must essentially be calcium-free (B-HTK, UW-CSS, COL) for it to be effective [20], as low extracellular sodium concentrations open calcium channels, which could increase cytosolic calcium and thus activate injurious reactions [21]. Thus acalcemia appears to be a prerequisite for an intracellular formulation. However, in extracellular-formulated cardioplegic solutions acalcemia is reported to be harmful by predisposing to the calcium paradox [20], a phenomenon of severe cellular damage that occurs when hearts are reperfused with a fluid containing calcium ions after a period of calcium-free perfusion [22]. Furthermore, this phenomenon is not specific to the myocyte as it has been noted in canine kidney perfusions, where it produced changes to the vessel walls and cell membranes [23].

Although we demonstrated a beneficial effect of an acalcemic ST solution on endothelial cells at hypothermia, this solution was possibly not truly acalcemic as it probably contained trace amounts of calcium as mentioned previously. However, survival decreased at normothermia with the acalcemic ST in contrast to ST, possibly as the likelihood of the calcium paradox increases with high extracellular sodium concentration and elevated temperature [24].

Chloride

We have shown that the low chloride content of intracellular solutions is an important feature in their composition, as Incell-High Cl, which had a higher chloride concentration than COL, resulted in diminished hypothermic endothelial cell survival. However, at normothermia Incell-High Cl produced better endothelial cell survival (51%) than the other intracellular solutions (<20%). Thus a low chloride content was beneficial at *hypothermia* in terms of endothelial cell survival and was associated with diminished morphological alterations. In contrast, at *normothermia* a low chloride concentration appeared to contribute to the observed cytotoxicity of the intracellular solutions.

In the extracellular solution ST, a low chloride concentration improved endothelial cell survival at hypothermia (71%) and was not detrimental at normothermia. However, the improved hypothermic endothelial cell survival provided by ST-Low Cl might have been partly due to a lower free ionized calcium as a result of calcium binding by gluconate [25]. Nevertheless, ST-Ca 0.05 mmol/L and ST-Ca 0.6 mmol/L, with free ionized calciums of less than 0.05 mmol/L and 0.5 mmol/L, respectively, thus bracketing the free calcium content of ST-Low Cl (free Ca^{2+} = 0.16 mmol/L), did not themselves significantly improve ST.

Chloride is a freely permeable extracellular anion, and ischemia-induced intracellular acidosis or elevated sodium level will encourage chloride to enter the cell, down its electrochemical gradient [26]. This entry would promote intracellular edema, as impermeate intracellular anions cannot leave the cell. Decreasing the chloride content of organ preservation solutions, by replacement with high-molecular-weight impermeate anions, helps regulate cellular edema [27]. Furthermore, these anions may bind calcium and magnesium and thus alter their free ionized concentrations, as we have demonstrated. Nevertheless a low chloride concentration was beneficial in both intracellular and extracellular solutions.

Additives: Buffers, Substrates

To determine why B-HTK was superior to the other intracellular solutions we examined No-HTK, a basic electrolyte formulation without the additives of B-HTK (histidine, tryptophan, KH-2-oxygluterate). This was inferior to B-HTK, and no better than COL or UW-CSS at both 24 hours and 36 hours after exposure. The beneficial effect of the HTK additives might have been wholly or in part due to the greater buffering capacity of histidine, as endothelial cells are known to be high lactate producers [1].

Potassium

The No-HTK solution has a potassium concentration of 10 mmol/L, in contrast to the otherwise similar COL solution (potassium concentration, 115 mmol/L) (see Tables 1, 2). Because both solutions gave similar results, we infer that the high potassium concentration of 115 mmol/L in the intracellular COL solution did not contribute to hypothermic cytotoxicity. However, a high potassium concentration has been reported to be harmful in extracellular solutions [25]. Furthermore, the observed cytotoxicity of the standard intracellular preservation solutions at normothermic exposure was not wholly due to a high potassium level, as B-HTK (normothermic survival <20%) only contains a potassium concentration of 10 mmol/L.

Temperature

In a previous study ST solution and the original Bretschneider-Procaine solution showed similar cytotoxicity, which increased equally when the temperature was raised from 19°C to 37°C [6]. However, this study evaluated endothelial cell suspensions by a method of dye exclusion (trypan blue). Furthermore, the Bretschneider-Procaine solution differs from the B-HTK solution now used internationally and in our study. In contrast, we show increased survival and adherence of the cells at normothermic (as opposed to hypothermic) exposure with ST. However, this increase might have been due to the improved buffering provided by the 5% CO_2 atmosphere used in all the normothermic groups.

As discussed in this report, the observed normothermic cytotoxicity of the intracellular solutions was related more to a low concentration of sodium, as well as a low concentration of chloride, than to a high concentration of potassium. These observations may be important when considering using cardioplegic solutions as reperfusion solutions at 37°C.

Conclusion

This study used a basic index, morphology and cell survival, to assess the endothelial response to preservation solutions, and more sensitive endothelial functions should also be assessed even if indirectly [7, 8], to highlight differences between less toxic solutions. Furthermore, although we do not suggest altering the composition of cardioplegic solutions on the basis of these findings, in a similar endothelial study assessing cardioplegic solutions used in South Africa [28], we showed poor endothelial preservation with some cardioplegic solutions being used clinically. This in turn correlated with another study that assessed postischemic myocardial function in an in vivo baboon heart model after protection with similar cardioplegic solutions [29]. We did not examine blood cardioplegia, which is not routinely used for long-term organ storage, because of the possibility of immunological incompatibility if not autologous. However, Harjula and associates [30] showed good endothelial preservation after 2 hours of exposure to blood cardioplegia at 22°C.

The best available solution for hypothermic endothelial cell preservation is the B-HTK cardioplegic solution; however, it should not be used at normothermia. Its superiority at hypothermia appears to be due to the low chloride content of intracellular solutions and to the additives histidine, tryptophan, and KH-2-oxygluterate in B-HTK. Furthermore, we were able to improve the efficacy of the extracellular ST cardioplegic solution in terms of endothelial cell survival by replacing chloride ions with gluconate.

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Role of Perfusion Pressure and Flow in Major Organ Dysfunction After Cardiopulmonary Bypass

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The role of perfusion pressure and flow during cardiopulmonary bypass with moderate hypothermia and hemodilution in the development of new postoperative renal or clinically apparent cerebral dysfunction was examined in 504 adults. Cardiopulmonary bypass flow was targeted at greater than 40 mL $\cdot$ kg^{-1} $\cdot$ min^{-1} and pressure at greater than 50 mm Hg. Flows and pressures less than target occurred in 21.6% and 97.1% of patients, respectively. Fifteen patients (3.0%) suffered new renal and 13 (2.6%) new central nervous system dysfunction. Low pressure or flow during cardiopulmonary bypass, expressed in absolute values or in intensity-duration units, were not predictors of either adverse outcome. Multivariate analysis identified use of postoperative intraaortic balloon counterpulsation ($p < 10^{-6}$), excessive blood loss in the ICU ($p < 10^{-4}$), need for vasopressors before cardiopulmonary bypass ($p < 10^{-4}$), postoperative myocardial infarction ($p < 10^{-3}$), emergency reoperation ($p < 0.002$), excessive postoperative transfusion ($p < 0.02$), and chronic renal disease ($p < 0.03$) as independent predictors of postoperative renal dysfunction. Independent predictors of postoperative central nervous system dysfunction were cardiopulmonary resuscitation in the intensive care unit ($p < 10^{-6}$), intracardiac thrombus or valve calcification ($p < 0.02$), and chronic renal disease ($p < 0.03$). Age greater than 65 years (40.7% of patients) did not predict either outcome. We conclude that failure of the native circulation during periods other than cardiopulmonary bypass rather than the flows and pressures considered here is the major cause of renal and clinically apparent central nervous system dysfunction after cardiac operations.

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Optimal outcome of cardiopulmonary bypass (CPB) includes uncompromised viability of the brain and kidneys after an unperfused heart protected by cardioplegia and hypothermia has been surgically treated. To achieve this outcome institutional guidelines for optimal perfusion flow and pressure vary widely and typically do not relate to degree of hypothermia or hemodilution employed. Recommended perfusion flows range from 30 to 60 mL $\cdot$ kg^{-1} $\cdot$ min^{-1} (1.2 to 2.4 L $\cdot$ m^{-2} $\cdot$ min^{-1}) and perfusion pressure ranges vary from 30 to 50 mm Hg to 60 to 100 mm Hg [1, 2]. Some outcome studies implicate hypotension during CPB as a cause of postoperative neurological (CNS) [3–6] and renal dysfunction [7–9], whereas others clearly fail to support these relationships [1, 10–15]. These reports, however, uniformly ignore the contribution of induced hypothermia to reduced tissue oxygen requirement and the contribution of hemodilution to the uncoupling of flow and pressure [16] in defining the role of hypotension or hypoperfusion in adverse outcome of CPB. Even though systematic review of these studies provides clear evidence of the importance of hemodilution in interpreting the role of hypotension, few reports describe either prime composition or hematocrit during CPB. As the surgical population undergoing CPB ages with an increasing incidence of preoperative cerebrovascular and renal disease, the role of CPB in the development of postoperative organ dysfunction continues as a timely issue because of questions that continue to arise when major organ dysfunction follows a cardiac operation. We therefore undertook a prospective investigation directed primarily to the roles of perfusion pressure and flow with hemodilution and moderate hypothermia in the genesis of new postoperative renal and clinically apparent CNS dysfunction in unselected adult patients undergoing operations requiring CPB. These data were then analyzed against the background of patient characteristics and other perioperative events that might also account for these adverse outcomes.

Material and Methods

With institutional review board approval granted in August 1988, 511 patients were prospectively studied from September 1, 1988, to March 31, 1989. All patients older than 19 years and undergoing elective operations requiring CPB were eligible for study. Patients were selected only on the basis of availability of an observer for record-

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ing data during operation. Only 2 patients were excluded, both because of preoperative end-stage renal disease. Study patients represented 28.8% of the 1,775 eligible adult patients operated on during this period. The distribution of operations among eligible and studied patients were, respectively, coronary artery bypass grafting (CABG), 75% and 68%; open chamber operations, 21% and 23%; and CABG with open chamber, 4% and 9%. Fifteen percent of eligible patients and 17% of studied patients had previous cardiac operations. All data were collected by observers not participating in patient care but trained to record events and hemodynamic data at specific intervals throughout operation.

Perioperative Management

Preoperative medication consisted of oral diazepam and intramuscular morphine or meperidine with scopolamine. Anesthesia was induced with either thiopental or a benzodiazepine and maintained with fentanyl, 10 to 50 μg/kg, and halothane, enflurane, or isoflurane. Muscle relaxation was provided by pancuronium. Ventilation was controlled to maintain arterial carbon dioxide tension at 35 to 40 mm Hg and arterial oxygen tension greater than 100 mm Hg. During operation bifrontal electroencephalogram, seven-lead electrocardiogram, direct radial artery blood pressure, and nasopharyngeal temperature were monitored continuously. Blood gas tensions corrected for temperature, plasma electrolytes, and hematocrit were measured intermittently.

All operations were performed through a median sternotomy. Several types of bubble or membrane oxygenators were used, each with a 40-μm arterial filter and cardiotomy suction filter. Oxygenators were primed with 20 mL/kg heparinized (2,000 U/L) dextrose 5% in lactated Ringer's solution. Heparin, 300 U/kg, was administered intravenously before cannulation for CPB and activated clotting time was always maintained greater than 350 seconds. During CPB, flow was targeted at 40 to 60 $\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ but exceeded or fell below this range when venous return varied. Mean arterial pressure (MAP) was targeted at 50 mm Hg but ranged from 20 to 100 mm Hg. During CPB, MAP was increased by bolus administration of phenylephrine or epinephrine or decreased by bolus administration of nitroglycerin or trimethaphan at the discretion of the anesthesiologist and surgeon. During CPB hematocrit was maintained between 0.18 and 0.29 with addition of red blood cells or crystalloid as necessary, and nasopharyngeal temperature between 28° and 32°C during the aortic cross-clamp period. Temperature-corrected carbon dioxide tension was maintained at 30 to 40 mm Hg and oxygen tension, greater than 150 mm Hg. Myocardial preservation included cold crystalloid cardioplegia and initial bathing of the heart in iced saline solution, which was removed from the pericardium before cardiac manipulation. Before termination of CPB air was aspirated by needle from the left ventricle and by continuous suction from the proximal aorta. To minimize the clinical consequences of cerebral emboli after open ventricle procedures, thiopental sufficient to establish an isoelectric electroencephalogram was given just before anticipated removal of the aortic cross-clamp [12]. No patient had absent electroencephalographic activity during any period of the operation except in response to thiopental. Urine output was monitored throughout operation, but no therapy was directed toward ensuring a minimal level of flow.

Outcome Indices

Adverse outcomes included new postoperative renal and clinically diagnosed neuropsychiatric dysfunction. Each was assessed 24 hours after completion of operation. When emergence from anesthesia was prolonged, neurologic evaluation was performed daily until a definitive outcome could be determined. Adverse CNS outcomes absent at the initial determinative postoperative evaluation and first appearing later in the postoperative period were not included. Renal dysfunction was considered present when the 24-hour postoperative creatinine level was greater than 130 μmol/L (1.5 mg/dL) and was at least 25% greater than the preoperative level. Urine output was assessed as a predictor of renal dysfunction but not as an outcome. New neuropsychiatric dysfunction was assessed by level of consciousness, orientation, movement of extremities in response to command, response to pin prick, tendon reflexes, and presence of abnormal behavior and ideation. These were compared with the preoperative examination. Preoperative and postoperative neurologic assessment was performed by physicians who did not participate in any other part of that patient's perioperative care or evaluation. Perioperative myocardial infarction (PMI), a secondary outcome unrelated to CPB pressure or flow, was also recorded. Diagnosis of PMI required a diagnostic change on the electrocardiogram (new Q wave or persistent left bundle-branch block) compared with the preoperative electrocardiogram and a 10-hour postoperative level of creatine kinase-MB greater than 80 IU [17].

Data Collection

Five observation periods were established: (1) pre-CPB, from induction of anesthesia to onset of CPB, (2) CPB, from onset of CPB to start of weaning, (3) weaning, the period 5 minutes before to 10 minutes after termination of CPB, (4) post-CPB, from weaning to end of operation, and (5) ICU, from arrival in the intensive care unit until 24 hours later. The CPB period was further divided into onset CPB, from initiation of CPB until perfusion pressure reached 50 mm Hg, and stable CPB, from end of onset CPB to start of weaning. Hemodynamic abnormalities during some periods were expressed in cumulative intensity-duration terms. During CPB, TM^{-50} was the product of the difference between 50 mm Hg and MAP less than 50 mm Hg and the minutes MAP remained less than 50 mm Hg [1, 3, 12] (Appendix 1). LF^{-40} was the product of the difference between perfusion flow of 40 $\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and lesser flows and minutes of flow less than 40 $\text{mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ (see Appendix 1). During pre-CPB, post-CPB, and weaning mean blood pressures in addition to systolic and diastolic were recorded. During these periods, $\text{TM}^{-0.7\text{PM}}$ was the product of the difference between 70% of the preoperative MAP and the observed MAP and the number of minutes MAP was less than 70% of preoperative MAP (see Appendix 1). Preoperative MAP was calculated from three to ten blood pressure determinations by nurses during preoperative hospitalization. During the ICU period hypotension was considered present when systolic blood pressure was less than 80 mm Hg for more than 15 minutes. This definition was chosen because of its common use to define hypotension during the postoperative period. Additional data recorded by observers included use of vasoactive drugs during or after operation, use of circulatory assist devices (intraaortic balloon pump [IABP] and Nimbus hemopump), urine output, emergency reoperation, ICU complications, and intraoperative and postoperative blood loss and transfusion.

Deaths

Four of the 511 patients studied died during operation. Two patients, 1 after redo CABG and 1 after aortic valve replacement, failed to be weaned from CPB despite IABP. Two others, 1 who refused transfusion and 1 with posterior ventricular rupture after mitral valve replacement, died of blood loss. Three patients required intraoperative Nimbus hemopump support after repeat CABG and failure to be weaned with IABP. One of these survived to hospital discharge. Data of these 7 patients were excluded because of absence of outcome data (4 patients) or the confounding effects of intraoperative circulatory failure on outcome measures (3 patients). All other patients survived the 24-hour ICU observation period.

Perioperative Myocardial Infarction

Fourteen patients (3.6% of CABG patients) suffered new PMI. All had undergone CABG with or without open chamber operations. Six had had previous CABG, 6 required IABP for weaning, 9 required vasopressor infusions during weaning and post-CPB, 5 suffered hypotension in the ICU, and 2 required cardiopulmonary resuscitation (CPR) in the ICU for ventricular fibrillation. All survived the observation period. Data from these patients were included.

Data Treatment

Continuous data, reported as mean ± standard deviation, were compared by two-tailed Student's t test and enumerative data by χ^2 corrected for continuity or by two-tailed Fisher's exact test, as appropriate. After first considering perfusion pressure and/or flow as independent predictors of adverse renal or CNS outcome, we determined univariately the influence of each patient characteristic and other perioperative observations on each adverse outcome. Characteristics and observations that were related to outcome at $p \leq 0.1$ as well as perfusion pressure and flow were entered into stepwise discriminant analysis using forward selection and backward elimination. A p value of less than 0.05 was required for entry and retention.

Table 1. Characteristics of All Patients and of Those With Postoperative Organ Dysfunction

Variable	All Patients	Patients With Dysfunction Renal	CNS
No. of patients	504	15	13
Age (y)	60.9 ± 11.0	65.3 ± 9.8	66.4 ± 11.0
Male (%)	72	67	69
Operations (n)			
CABG	345	11	7
CABG + open chamber	45	2	1
Open chamber	114	2	5
Previous cardiac operations (%)	17	27	31
Diseases present before operation (%)			
Renal[a]	8.7	40[f]	31[g]
Neurologic disease[b]	13.9	33[g]	23
Left ventricular dysfunction[c]	20.4	40	46[g]
Peripheral vascular disease[d]	9.5	27[g]	23
Diabetes[e]	20.0	47[g]	31
Chronic hypertension	44.6	27	46

[a] Creatinine level ≥ 130 μmol/L (1.5 mg/dL) and/or renovascular disease documented by arteriography. [b] History of transient ischemic attack or stroke and/or cerebrovascular disease documented by arteriography. [c] Left ventricular end-diastolic pressure ≥ 20 mm Hg (before contrast ventriculography) or left ventricular ejection fraction ≤ 0.40 and/or documented history of congestive heart failure. [d] Documented by angiography and/or exercise induced claudication. [e] Insulin- and non-insulin–dependent. [f] $p < 0.001$. [g] $0.001 < p < 0.05$ by univariate analysis compared with 478 unafflicted patients in the first column.

CABG = coronary artery bypass grafting; CNS = central nervous system.

Results

Five hundred four patients completed the study, of whom 15 suffered new perioperative renal and 13 new CNS dysfunction. Two patients suffered both renal and CNS dysfunction and were included in both groups.

Patient Characteristics

By univariate analysis patients with new postoperative renal dysfunction had significantly higher incidences of preoperative renal and CNS dysfunction, as well as peripheral vascular disease and diabetes, than did the patients without postoperative renal dysfunction (Table 1). New postoperative CNS dysfunction was significantly associated only with preoperative renal and left ventricular dysfunction. Chronic hypertension did not increase risk of either outcome. Study patients ranged in age from 19 to 89 years with 205 patients (40.7%) aged 65 years or greater. Patients with adverse outcomes tended to be older than those without but the differences were not significant (see Table 1). Preoperative chronic use of

Table 2. Mean Arterial Pressure and Duration-Intensity Indices of Hypotension During Perioperative Periods

Variable	No Dysfunction	Renal Dysfunction	CNS Dysfunction
No. of patients	478	15	13
Pre-CPB[a]			
Low MAP[b] (mm Hg)	61.6 ± 9	54.3 ± 10[e]	59.1 ± 11
TM$^{-0.7\text{PM}}$[c] (mm Hg · min)	113 ± 326	145 ± 167	117 ± 185
CPB[a]			
Onset TM^{-50}[c] (mm Hg · min)	152 ± 218	214 ± 246	121 ± 203
Stable TM^{-50}[c] (mm Hg · min)	91 ± 179	161 ± 177	113 ± 126
Weaning[a]			
Low MAP[b] (mm Hg)	65.6 ± 13	56.9 ± 13[e]	63.8 ± 15
TM$^{-0.7\text{PM}}$[c] (mm Hg · min)	46 ± 86	92 ± 205	60 ± 77
Post-CPB[a]			
Low MAP[b] (mm Hg)	66.1 ± 11	59.6 ± 9[e]	64.3 ± 9
TM$^{-0.7\text{PM}}$[c] (mm Hg · min)	54 ± 163	83 ± 152	57 ± 80
ICU[a] Hypotension[d] (%)	11.1	47[f]	31[e]

[a] See Material and Methods section for definition of periods. [b] Mean of the lowest single mean arterial pressure (MAP) values recorded during the observation period. [c] See Appendix 1. [d] Systolic blood pressure less than 80 mm Hg for more than 15 minutes in ICU. [e] $0.001 < p < 0.05$ compared with no-dysfunction group by univariate analysis. [f] $p < 0.001$ compared with no-dysfunction group by univariate analysis.

CPB = cardiopulmonary bypass; CNS = central nervous system; ICU = intensive care unit.

nitrates, β-adrenergic blockers, calcium entry blockers, digoxin, diuretics, or other antihypertensive drugs was not related to outcome.

Cardiopulmonary Bypass Variables

As expected when using a crystalloid prime, virtually all patients (97.1%) had perfusion pressures of less than 50 mm Hg during onset CPB. During stable CPB, MAP less than 50 mm Hg occurred in 59.9% of patients and its occurrence was not related to either adverse outcome. In 24 patients perfusion pressure was less than 50 mm Hg throughout CPB; none suffered postoperative organ dysfunction. Cardiopulmonary bypass hypotension, expressed as TM^{-50}, during onset CPB and stable CPB was also not related to either adverse outcome (Table 2). Flow rate during stable CPB was less than 40 mL · kg^{-1} · min^{-1} in 21.6% of all patients and was not more common in those with renal (27%) or CNS (15%) dysfunction. When expressed as both degree and duration, LF^{-40} was not significantly associated with either adverse outcome (Table 3). By univariate analysis, new renal dysfunction was associated with longer duration of CPB, but neither adverse outcome was related to duration of aortic cross-clamp, pressor administration, or low urine output (<0.5 mL · kg^{-1} · hr^{-1}) during CPB (see Table 3). Use of vasodilators or highest perfusion pressure during CPB were not predictive of adverse outcome.

Table 3. Perioperative Observations in Patient Groups With and Without Postoperative Organ Dysfunction

Variable	No Dysfunction	Renal Dysfunction	CNS Dysfunction
Pre-CPB pressors[a] (%)	0.8	20[d]	8
CPB			
Pressor administration[b] (%)	69.1	67	54
Urine output < 0.5 mL · kg^{-1} · h^{-1} (%)	9.3	20	8
Duration (min)	60.1 ± 26	76.2 ± 27[e]	62.0 ± 21
Aortic clamp time (min)	38.8 ± 18	46.1 ± 27.5	41.9 ± 14
Integrated low flow (LF^{-40})[c] (mL/kg/min · min)	13.7 ± 50	26.7 ± 62	18.8 ± 56
Weaning (%)			
Pressor infusion[a]	8.8	40[d]	23
Intraaortic balloon pump	4.2	13	8
Post-CPB pressors[a] (%)	12.8	47[d]	38[e]
Operative (%)			
Blood loss ≥1,000 mL	21.3	53[e]	31
Transfusion ≥750 mL	21.8	60[e]	23
Intracardiac clot or calcium	7.7	0	23[e]
ICU (%)			
Hypotension	11.1	47[d]	31[d]
Pressor infusion[a]	15.7	53[d]	54[d]
Intraaortic balloon pump	0	27[d]	8
Cardiopulmonary resuscitation	0.8	27[d]	31[d]
Reoperation	3.8	40[d]	15
Blood loss ≥1,500 mL	14.6	73[d]	31
Transfusion ≥1,000 mL	20.9	67[d]	38
Perioperative myocardial infarction	1.8	20[d]	8

[a] Infusion of dopamine, dobutamine, amrinone, epinephrine, or norepinephrine. [b] Bolus administration of phenylephrine or epinephrine. [c] See Appendix 1. [d] $p < 0.001$ compared with no dysfunction group. [e] $0.001 < p < 0.05$ compared with no dysfunction group.

CPB = cardiopulmonary bypass; CNS = central nervous system; ICU = intensive care unit.

Blood Pressure During Unassisted Circulation

Patients who suffered renal but not CNS dysfunction had significantly lower MAP (single observations) during the pre-CPB, weaning, and post-CPB periods (see Table 2). However, hypotension over time expressed as TM$^{-0.7\text{PM}}$

Table 4. Perioperative Characteristics[a] Independently Predicting Postoperative Renal and Neurological Dysfunction Identified by Discriminant Analysis

Characteristic	F Value[b]
Renal dysfunction	
ICU intraaortic balloon pump	68.1
ICU blood loss ≥ 1,500 mL	15.5
Pre-CPB pressors	15.4
Perioperative myocardial infarction	11.4
Emergency reoperation	9.4
ICU transfusion ≥1,000 mL	5.9
Preop renal disease	5.4
CNS dysfunction	
ICU cardiopulmonary resuscitation	50.6
Intracardiac clot or calcium	5.4
Preop renal disease	4.9

[a] All characteristics different between no-dysfunction and either dysfunction group at $p \leq 0.1$ were included in this analysis. [b] F values quantitatively describe the power of the independent relationship of each characteristic retained in the discriminant analysis by virtue of a p value < 0.05. In this analysis an F value of 4.9 indicates a statistical probability of 0.028; an F of 68.1 indicates a probability of $<10^{-6}$.

CPB = cardiopulmonary bypass; CNS = central nervous system; ICU = intensive care unit.

during any of these observation periods was not significantly different. Both renal and CNS dysfunction were more likely to occur in patients who suffered hypotension during the ICU period (see Table 2).

Perioperative Variables

Patients who suffered new perioperative renal dysfunction were more likely to receive vasopressors by infusion to treat hypotension during all periods except CPB, bled greater amounts and received more blood products during operation, and more frequently required circulatory support by IABP. They also suffered more bleeding and hypotension in the ICU period requiring pressors, greater transfusion, CPR, and reexploration for bleeding (see Table 3). Perioperative myocardial infarction occurred in 20% of patients with renal dysfunction as compared with 1.8% of those without. Patients who experienced new perioperative CNS dysfunction were more likely to have calcified heart valves or intracardiac clot at operation, to receive pressor infusions during the post-CPB and ICU periods, and to require CPR in the ICU (see Table 3). Perioperative use of diuretics, hypertension, or use of vasodilators during perioperative periods other than CPB were not predictive of adverse outcome.

Discriminant Analyses

All patient characteristics and perioperative variables that differed between patients with and without adverse outcomes at $p \leq 0.1$ and CPB perfusion pressure and flow data were subjected to discriminant analysis. Factors that were independently associated with the outcome were determined (Table 4). The strongest predictors of renal dysfunction related to hemodynamic instability after weaning from CPB secondary to either cardiac dysfunction or bleeding, the occurrence of PMI, and the presence of preoperative chronic renal disease. The factors independently associated with postoperative CNS dysfunction were CPR in the ICU, presence of intracardiac debris at operation, and preoperative renal disease. Cardiopulmonary bypass pressure and flow were not predictive. However, because two events unrelated to CPB characteristics, CPR in the ICU and PMI, were such strong predictors of adverse outcomes, we considered the possibility that inclusion of patients with these events masked a significant effect of CPB pressure and flow. Discriminant analysis was therefore repeated excluding the 11 patients who required CPR in the ICU, repeated again without the 14 patients who suffered PMI, and repeated eliminating both. None of these additional analyses unmasked a significant relationship between any CPB characteristic and either adverse outcome by univariate or discriminant analysis.

Clinical Analysis of Adverse Outcomes

In 2 patients both CNS and renal complications developed. Both underwent uneventful operations, one for replacement of the aortic valve and ascending aorta and the other for CABG. Both suffered severe bleeding in the ICU leading to CPR and reoperation. One subsequently had marked brainstem dysfunction with mild azotemia and died. The other sustained hemiparesis, with a peak creatinine level of 350 µmol/L (4 mg/dL) and left the hospital with minimal gait dysfunction and normal renal function.

Of the remaining 11 patients with postoperative CNS dysfunction, five underwent CABG and 6, open ventricle operations. Of the 5 CABG patients, 1 required IABP and pressors for weaning. Two had severe bleeding in the ICU requiring blood transfusions of more than 1,000 mL, pressors, and CPR in 1. The other suffered PMI. The fourth patient had an uneventful operation 2 weeks after myocardial infarction but awakened with disorientation and weakness of the right leg. A mural thrombosis was suspected. Computed tomographic scan confirmed a left posterior cerebral infarction. A fifth patient with preoperative syncopal attacks of unknown etiology and hemodynamic instability in the pre-CPB period had an uneventful operation but awoke with complete left hemiparesis and confusion. Of the 6 patients with open ventricle operations, 3 had calcified aortic valves replaced and each suffered focal neurological deficits. The fourth patient, who had aortic valve replacement for endocarditis, suffered severe bleeding with hypotension in the ICU. The fifth patient, who had chronic atrial fibrillation, underwent replacement of a mitral prosthesis and wakened with disoriention and left leg paralysis. The last patient underwent uneventful aortic valve replacement and awakened with disoriention and lethargy, which improved slowly. Of these 11 patients, postoperative CNS dysfunction in 9 could be reasonably attributed to embolism or to acute circulatory failure, cardiac or hemorrhagic, unrelated to CPB characteristics. In the 2 patients without suspected embolism or circulatory failure, CPB durations were 49 and 50 minutes, lowest CPB perfusion pressures were 40 and 50 mm Hg, TM^{-50} were 10 and 0 mm Hg · min, and LF^{-40} were 0 and 0 mL/kg/min · min, respectively. Eight of these 11 patients were either normal or improving on discharge from the hospital.

Of the 13 additional patients with postoperative renal dysfunction, 10 underwent CABG. Four required IAPB, pressors, and transfusion for weaning, including 2 who required CPR in the ICU. Four other patients had major bleeding during and after operation, requiring transfusion, pressors, and in 1, CPR in the ICU. Two patients who underwent uneventful CABG demonstrated a 24-hour increase in creatinine level from 100 to 150 μmol/L (1.1 to 1.7 mg/dL) and 110 to 150 μmol/L (1.2 to 1.7 mg/dL), respectively. Of the 3 patients with open ventricle operations, 2 suffered postoperative renal dysfunction after aortic valve replacement plus CABG. Both had mild preoperative renal dysfunction and suffered hypotension requiring large transfusions and pressors in the ICU. One required CPR in the ICU, the other suffered PMI. The last patient, who had donated a kidney several years previously, required pressor infusions for several hours after weaning and transfusions in excess of 6 units in the ICU. Of the 13 patients with postoperative renal dysfunction, all but 2 suffered major episodes of acute circulatory failure unrelated to CPB, which could reasonably account for postoperative dysfunction. In the 2 patients without circulatory failure, CPB durations were 132 and 82 minutes, lowest CPB perfusion pressures were 40 and 40 mm Hg, TM^{-50} were 60 and 110 mm Hg · min, and LF^{-40} were 25 and 0 mL/kg/min · min, respectively. Eleven of the 13 patients with renal dysfunction only left the hospital with normal creatinine levels and without need for dialysis.

In no patient without renal or CNS dysfunction at the initial definitive evaluation did major organ dysfunction subsequently develop during hospitalization.

Comment

A number of studies describe a clear association between low perfusion pressure and CNS [3–6] and renal [7–9, 18] dysfunction after operations requiring CPB. For example, Stockard and associates [3] described severe neurological deficits in 6 of 7 patients with TM^{-50} greater than 100 mm Hg · min compared with 3 of 16 when TM^{-50} was less than 100 mm Hg · min. Javid and co-workers [6] reported neuropsychiatric dysfunction in 41 of 100 patients and associated it with duration of perfusion pressure less than 60 mm Hg and absolute CPB pressure less than 50 mm Hg. Postoperative renal dysfunction was described by Bhat and associates [7] in 150 of 490 patients as significantly related to duration of perfusion pressure less than 60 mm Hg. Interpretation of these data require caution, because hypotension occurring during periods other than CPB were not similarly considered. More importantly, these observers either used a blood prime in the oxygenator or did not report the degree of hemodilution employed and assumed that low perfusion pressure, as in the natural circulation, reflected low perfusion flow. The use of hemodilution with crystalloid as primary or total oxygenator prime provides a perfusate of much lower viscosity which, based on purely physical principles, causes perfusion pressure to be lower at the same perfusion flow without any change in arteriolar resistance. With perfusion techniques unchanged and the advent of hemodilution, hypotension during CPB no longer reflected low flow, only acutely reduced perfusate viscosity [16]. Although more recent data attest to the safety of lower pressures and flows when employing hemodilution and hypothermia [1], many institutions apparently retain their traditional postures with regard to minimal pressures and flows. Most publications reporting use of CPB continue to report perfusion pressures or flows without perfusate hematocrit with which perfusate viscosity varies directly. The primary purpose of this study was to examine the safety of the pressures and flows observed during CPB with a low-viscosity perfusate and to determine their role in postoperative CNS and renal dysfunction.

To study this question in humans neither low pressure nor flow could be ethically induced. Rather, as typical of most perfusions, variable periods of reduced flow and pressure occurred in almost all patients when venous return, viscosity, vascular resistance, and temperature changed deliberately or inadvertently. Not surprisingly, absolute levels of lowest perfusion flow and pressure were not related to any adverse outcome as most episodes were transient. If, however, a relationship existed between CPB flow or pressure and adverse outcomes, it should be revealed by quantification of duration and intensity as attempted here. We could identify no relationship. By contrast a clear relationship existed between adverse outcome and evidence of hypoperfusion during natural circulation in the pre-CPB, post-CPB, and ICU periods. Of seven factors considered independently predictive by discriminant analysis of postoperative renal dysfunction, three reflect cardiac-related hypoperfusion (pre-CPB pressors, ICU IABP, and PMI) and three hemorrhage-induced hypoperfusion (volume of ICU blood loss and transfusion and emergency reoperation). Of three factors independently predictive of postoperative CNS dysfunction, one related to postoperative hypoperfusion (ICU CPR) and one to potential embolization (calcium/clot in ventricle). Preoperative renal disease predicted both adverse outcomes. A reason for its association with adverse CNS outcome is unclear, but the relationship has been noted previously [1].

For postoperative renal dysfunction, our data confirm a lack of association with CPB perfusion characteristics [14] or urine output during CPB [15] but a strong relationship with markers of poor postoperative cardiac function [14, 15] and preexisting renal dysfunction [14, 15, 19]. Similarly our univariate predictors confirm observations suggesting a strong relationship with existing vascular disease [9]. We were able to establish these relationships using a relatively insensitive measure of postoperative renal dysfunction, the 24-hour serum creatinine value.

This marker was chosen because of detectability in the early postoperative period, reasonable assurance of a temporal association with operation and CPB, and a reasonable assurance that the magnitude of change was clinically important. Although blood urea nitrogen and serum creatinine levels are generally crude measures of renal function, Charlson and co-workers [19] confirmed a 50% decrease in creatinine clearance when serum creatinine measured 24 hours postoperatively increased by the criteria used in this study. The test had reasonable sensitivity in this study because no patient diagnosed as without renal dysfunction on initial evaluation had renal dysfunction during subsequent hospitalization. We found post-CPB and ICU oliguria to be poor markers of incipient renal dysfunction and attributed that to the widespread treatment of oliguria with potent diuretics and "renal-dose" dopamine.

Central venous system dysfunction after operations requiring CPB does not have a single cause. Gross neurological deficits and psychiatric abnormalities of the type described here primarily result from either embolism or hypoperfusion from episodes of circulatory failure occurring during periods other than CPB. When sought prospectively, clinically apparent dysfunction is present on emergence from anesthesia in 2.9% to 16.2% of patients after CPB [1, 12, 13]. Any neuropsychiatric dysfunction directly related to CPB should be apparent at this time [12, 13]. High frequencies of psychometric deterioration without clinically detectable neuropsychiatric abnormalities have been reported after CPB [20, 21] and may relate to microembolization of air or particulate matter and duration of CPB. Although it is widely assumed that decrements in psychometric performance after CPB are lesser manifestations of the same process leading to clinically detectable neuropsychiatric deficits, this relationship is not well supported. Decrements in psychometric performance do not correlate specifically with known predictors of clinically apparent CNS deficits such as open ventricle operations and calcified valves [13, 22]. Further, these decrements in performance occur postoperatively with high frequency in operations that do not involve the heart or great vessels and are rarely associated with clinical CNS dysfunction [23]. For these reasons psychometric testing was not part of this investigation.

The absence of an association between CPB flow or pressure and clinically apparent postoperative CNS dysfunction observed in this study is in consonance with all reports in which hemodilution was used during perfusion [1, 10–13, 24]. Govier and associates [11] demonstrated remarkable preservation of regional cerebral blood flow and autoregulation in 67 patients during perfusions using a flow of 1.6 $L \cdot min^{-1} \cdot m^{-2}$ and pressure of 30 mm Hg during hypothermia of 25° to 29°C. In no patient in our study were the observed flows and pressures associated with global cerebral ischemia as monitored by bifrontal electroencephalogram. The remarkable preservation of cerebral blood flow during CPB applies equally to the elderly despite the fact that neuropsychiatric complications follow CPB almost five times as frequently in patients aged greater than 60 years [13]. Brusino and colleagues [25] compared cerebral blood flow in groups of patients aged less than 50 years and greater than 65 years during hypothermic CPB at a flow rate of 1.6 $L \cdot min^{-1} \cdot m^{-2}$ and an average perfusion pressure of 43 ± 21 mm Hg in the group aged more than 65 years. Autoregulation was not impaired in either group and cerebral blood flow was not different between them. In 421 patients who underwent CABG with hemodilution to a hematocrit of 0.20 ± 0.04, Breuer and associates [26] also found CNS dysfunction after CPB related to markers of postbypass cardiac dysfunction but not related to CPB perfusion flow or perfusion pressure or age of the patient. Forty-nine (9.7%) of our patients were 75 years old or older and 205 (40.7%) were 65 years old or older. Eighteen (69%) of the 26 patients who experienced either renal or CNS dysfunction were 65 years old or older ($p < 0.005$). Although the degenerative diseases of the aged were related to adverse outcomes (see Table 1), age itself was not a significant independent predictor of postoperative major organ dysfunction.

A recent similar study by Shaw and colleagues [27] addressed the same question as this study but was limited to CNS dysfunction in patients undergoing CABG only. Outcome data included changes in a battery of ten psychometric tests and visual acuity, and retinal examinations, as well as daily neurological examination. Fifty patient variables drawn from the preoperative, intraoperative, and postoperative periods were examined as predictors of postoperative CNS dysfunction by discriminant analysis. In their patients mean perfusion pressure less than 40 mm Hg for more than 10 minutes was not a predictor of the types of CNS dysfunction we observed.

We conclude that during hypothermic CPB with hemodilution, perfusion flow and radial artery pressure in the ranges described here including periods when targeted values were not maintained are unrelated to new neurological or renal dysfunction after CPB and are safe even for elderly patients undergoing cardiac operations. The traditional indictment of inadequate cerebral perfusion based on an observed perfusion pressure less than 50 mm Hg during CPB is not valid when hemodilution is employed with uncoupling of pressure-flow relationships by low viscosity. Both adverse outcomes related most strongly to hypoperfusion during failure of the native circulation in the period after CPB and the early postoperative period.

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Appendix 1

The following formulas were used for calculating less than targeted cardiopulmonary bypass flow and pressure intensity-duration integrations. The formulas apply to each episode of less than targeted values. The final value is the sum of all episodes. (MAP = mean arterial blood pressure.)

$$\mathrm{TM}^{-50} = \Sigma\,[50\text{ mm Hg} - (\text{lowest MAP} < 50\text{ mm Hg})] \times \text{duration (min) of MAP} < 50\text{ mm Hg}$$

$$\mathrm{TM}^{-0.7\mathrm{PM}} = \Sigma\,[70\text{ mm Hg} - (\text{lowest MAP} < 70\%\text{ average preoperative MAP})] \times \text{duration (min) of MAP} < 70\%\text{ average preoperative MAP}$$

$$\mathrm{LF}^{-40} = \Sigma\,[40\text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1} - (\text{lowest CPB flow rate} < 40\text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1})] \times \text{duration (min) of CPB flow} < 40\text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$$

Long-Term Results of Operation for Non–Small Cell Lung Cancer in the Elderly

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We surgically treated 185 patients with non–small cell lung cancer who were 70 years old or older. The operative mortality rate was 3%, and the 5-year survival rate was 48%. The mortality and prognosis were similar to those in younger patients. The number of elderly patients who smoked heavily or who had ventilatory defects was high, but the incidence of pneumonectomy was low. There were no differences based on age in regard to histological type, TNM classification, and curability. Pulmonary complications occurred in 21% of the elderly patients and were correlated with preoperative pulmonary function and smoking habits. When the elderly are to undergo elective pulmonary resection for lung cancer, the preoperative evaluation of pulmonary function should be thorough, and both preoperative and postoperative physical therapy should be given. If postoperative pulmonary function is predicted to be less than 0.8 L/m^2 of vital capacity and 0.6 L/m^2 of forced expiratory volume in 1 second, a limited resection or nonsurgical therapy should be considered.

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In 1989, we [1] reported that bronchogenic carcinoma occurs mainly in patients of either sex in the seventh decade of life. On the other hand, the life expectancy in Japan, as of 1988, was advanced from age 70 years to about 82 years for men and about 85 years for women; the corresponding increases from the age of 80 years are 6.69 and 8.36 years [2]. The number of patients 70 years of age or older with lung cancer is expected to increase, and these patients represent a high risk for pulmonary operation [3]. Long-term survival rates after resection for lung cancer in elderly patients have been reported, but the associated mortality rates range from 14% to 20% [4–7].

This report assesses preoperative findings, postoperative complications, mortality, and late results of operation for non–small cell lung cancer in elderly patients compared with findings in younger patients treated during the same period.

Material and Methods

During the 15-year period 1974 to 1989, 662 Japanese patients with histologically proven non–small cell lung carcinoma were surgically treated in the Department of Surgery II, Faculty of Medicine, Kyushu University. Of these patients, 185 (28%) were 70 years of age or older. The findings in this older group were compared with the findings in the 477 younger patients.

All records for each patient were reviewed for age at the time of operation, sex, use of tobacco, pulmonary function test results, histological type, TNM classification, stage, type of operation done and curability, postoperative complications, postoperative death, and 5-year survival.

Smoking history was based on the index of Brinkman and Coates [8], that is, the sum of the number of cigarettes smoked per day multiplied by the years of smoking. Pulmonary function tests included measurement of vital capacity (VC) and its first-second forced expiratory volume (FEV$_1$) and were performed using a spirometer [9]. The test results were evaluated as follows: normal, %VC greater than 80 and %FEV$_1$ greater than 70; restrictive, %VC less than or equal to 80; obstructive, %FEV$_1$ less than or equal to 70; and combined ventilatory defects, %VC less than or equal to 80 and %FEV$_1$ less than or equal to 70 [10]. The patients with ventilatory defects were routinely treated preoperatively and postoperatively with a device with increased dead space and expiratory pressure (IDSEP breather [Miconn Co, Inc, Upper Darby, PA] or Souffle [Kayaku Co, Ltd, Tokyo, Japan]) [11].

To determine which patients would be safe candidates for pulmonary resection, the loss of pulmonary function was calculated by the method of Ali and associates [12] or was estimated from a direct reading of radionuclide scans [13]. Based on these predictions of postoperative pulmonary function and using a VC value of 0.8 L/m^2 or greater and an FEV$_1$ value of 0.6 L/m^2 or greater [14, 15], the patients were treated by complete resection, that is, lobectomy or pneumonectomy combined with mediastinal lymph node dissection, whenever possible. If the values were lower than these, the patients were treated by wedge or segmental resection or were considered to have inoperable disease. Incomplete resection was defined as pulmonary resection and lymph node dissection that did not eradicate the tumor.

Resected specimens were routinely examined patholog-

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Table 1. Distribution of 662 Patients by Age and Sex

Age (y)	Male	Female	Total
≤29	1	0	1
30–39	11	4	15
40–49	29	13	42
50–59	129	46	175
60–69	183	61	244
70–79	137	33	170
≥80	8	7	15
Total	498	164	662

ically and reviewed using the current criteria of the World Health Organization [16]. The postsurgical (pathological) stages were determined according to the TNM classification of the Union Internationale Contre le Cancer [17].

Survival curves were calculated using the Kaplan-Meier life-table procedure [18], and the significance of difference was analyzed by the generalized Wilcoxon test [19].

Results

The age distribution of the patients is shown in Table 1. The peak incidence of lung cancer occurred in the 60 to 69-year-old group. Elderly patients were defined as patients 70 years old or older. Of the 662 patients studied, there were 185 elderly patients (28%), 145 men and 40 women. The male to female ratio did not differ significantly in each age group.

In the elderly group, 147 patients (79%) were cigarette smokers; of these, 53 (36%) had a Brinkman index of greater than 1,201. Pulmonary function studies showed 110 patients (59%) with a normal pattern, 31 (17%) with a restrictive pattern, 32 (17%) with an obstructive pattern, and 11 (6%) with combined ventilatory defects (Table 2). The number of patients who smoked heavily and had pulmonary dysfunction was higher in the elderly patients compared with the younger patients.

The most common histological types in the elderly group were adenocarcinoma (46%) and squamous cell carcinoma (42%), followed by large cell carcinoma and adenosquamous carcinoma. There was no significant difference between the elderly and younger groups in regard to histological type.

Table 2. Results of Pulmonary Function Tests[a]

Type of Ventilatory Defect	Elderly Patients	Younger Patients	Total
Normal	110 (59)[b]	333 (70)	443 (67)
Restrictive	31 (17)	57 (12)	88 (13)
Obstructive	32 (17)	65 (14)	97 (15)
Combined	11 (6)	19 (4)	30 (5)
Not examined	1 (1)	3 (1)	4 (1)
Total	185	477	662

[a] Numbers in parentheses are percentages. [b] Significance: $p < 0.01$ between the two groups.

Table 3. Staging[a]

Stage	Elderly Patients	Younger Patients	Total
I	96 (52)	204 (43)	300 (45)
II	8 (4)	32 (7)	40 (6)
IIIA	46 (25)	123 (26)	169 (26)
IIIB	22 (12)	51 (11)	73 (11)
IV	6 (3)	32 (7)	38 (6)
Unclassified	7 (4)	35 (7)	42 (6)

[a] Numbers in parentheses are percentages.

Patient staging was evaluated by age group (Table 3). There were no significant differences between the two age groups. The most frequently performed operation in both age groups was lobectomy (Table 4). Pneumonectomy was done in 6% of patients in the elderly group, an incidence significantly lower than in the younger group. In the elderly group, 139 patients (75%) underwent complete resection, 28 (15%) had incomplete resection, and 18 (10%) were treated by exploratory thoracotomy only. There was no significant difference between the two age groups in regard to curability of the operation.

The postoperative course in the elderly group was uneventful for 116 patients (63%). Sixty-nine patients (37%) had postoperative complications, pulmonary complications in 39 (57%) of them. However, there were no significant differences compared with the younger group (Table 5). There was a definite relationship between pulmonary function and smoking habits and the incidence of pulmonary complications.

Of the elderly patients with complications, 6 (9%) had major complications and died; 4 died within the first 30 postoperative days, and 2 died after that time. All 6 patients who died had undergone lobectomy; none had

Table 4. Type of Operation and Curability[a]

Variable	Elderly Patients	Younger Patients	Total
Operation			
Pneumonectomy	11 (6)[b]	80 (17)	91 (14)
Bilobectomy	10 (5)	40 (8)	50 (8)
Lobectomy	137 (74)	293 (61)	430 (65)
Partial resection	9 (5)	12 (3)	21 (3)
Exploratory thoracotomy	18 (10)	52 (11)	70 (10)
Curability			
Complete resection	139 (75)	338 (71)	477 (73)
Incomplete resection	28 (15)	85 (18)	113 (17)
Exploratory thoracotomy	18 (10)	54 (11)	72 (11)

[a] Numbers in parentheses are percentages. [b] Significance: $p < 0.01$ between the two groups.

had pneumonectomy because pneumonectomy was avoided in the elderly patients whenever possible. On the other hand, of the seven hospital deaths (1%) in the younger group, four occurred in patients who had had pneumonectomy, one occurred in a patient who had had bilobectomy, and two, in patients who had undergone lobectomy. The mortality was essentially the same in both groups.

The overall 5-year survival rate in the elderly patients was 48% and in patients less than 70 years old, 41% (Table 6). Based on stage, the 5-year survival rate for patients in stage I was 67% in the elderly group compared with 68% in the younger group and for stage IIIA, 32% in the elderly group compared with 30% in the younger group. There were no significant differences between the age groups.

Comment

Surgical intervention for lung cancer in the elderly is becoming more common as life expectancy increases. As only surgical treatment of lung cancer offers a hope of cure, the thoracic surgeon is asked to consider operative intervention in the elderly justified in carefully selected patients. The type of surgical treatment for lung cancer in the elderly depends on the general clinical condition of the patient and the stage of the disease. The major findings in the present study were that the survival and mortality rates and the incidence of postoperative complications in the older patients with non–small cell lung cancer were similar to those in the younger patients.

Table 5. Incidence of Complications and Hospital Mortality[a]

Variable	Elderly Patients (n = 184)	Younger Patients (n = 472)	Total (n = 656)
Uneventful course	116 (63)	325 (69)	441 (67)
Pulmonary complications	39 (21)	101 (21)	140 (21)
Atelectasis	10	30	40
Pneumonia	8	18	26
Prolonged air leak	17	42	59
Bronchopleural fistula	3	6	9
Thoracic empyema	1	3	4
ARDS	0	2	2
Cardiac-related complications	25 (14)	37 (8)	62 (9)
Arrhythmia	23	37	60
Myocardial infarction	1	0	1
Ruptured aortic aneurysm	1	0	1
Other complications	4 (2)	9 (2)	13 (2)
Gastrointestinal bleeding	0	5	5
Delirium	4	4	8
Hospital mortality	6 (3)	7 (1)	13 (2)
Pneumonia	3	2	5
Thoracic empyema	1	2	3
ARDS	0	1	1
Myocardial infarction	1	0	1
Ruptured aortic aneurysm	1	0	1
Gastrointestinal bleeding	0	2	2

[a] Numbers in parentheses are percentages. The course of 6 patients was unknown.

ARDS = adult respiratory distress syndrome.

Table 6. Survival Rates in Elderly and Younger Patients by Stage

Stage	Age Group	Survival rates (%) 1 Year	2 Years	3 Years	4 Years	5 Years	p Value
I	Elderly	95	93	78	70	67	0.1691
	Younger	95	87	79	73	68	
II	Elderly	67	40	40	40	40	0.9707
	Younger	90	62	43	31	17	
IIIA	Elderly	74	45	32	32	32	1.1426
	Younger	82	59	44	38	30	
IIIB	Elderly	52	39	39	39	39	0.5231
	Younger	53	31	18	13	13	
IV	Elderly	50	33	18	18	18	0.6951
	Younger	62	44	22	15	15	
Total	Elderly	82	68	55	50	48	0.6621
	Younger	80	63	52	46	41	

Yellin and Benfield [20] discussed four fundamental aspects of lung cancer in the elderly: the biological activity of the lesion, the life expectancy, the risk of thoracotomy, and the value of operation. They concluded that the most important life-limiting factor in elderly patients with lung cancer was a malignant lesion, not age. Other investigators [3–5] reported that the 5-year survival rate of patients 70 years old or older was about 30% and that the rates did not differ from those of younger patients. We found that the 5-year survival rate was 48% in the elderly patients and had expected the prognosis to be favorable.

The operative mortality rate reported ranges from 14% to 20% in patients 70 years old or older; higher rates have been noted for those treated by pneumonectomy [3–7]. The rate of postoperative complications in these older patients ranges from 18% to 37% [21, 22]. The mortality rate, however, is reported to be less than 7.1%, reflecting judicious selection of patients [23]. The choice of a limited operation for high-risk patients has led to a decrease in operative mortality [24]. Six of our older patients died of postoperative complications, and they were all heavy smokers with a Brinkman index greater than 801. The incidence of pulmonary complications in the elderly patients in this study was 21%; these complications included prolonged air leak (longer than 7 days), atelectasis, pneumonia, bronchopleural fistula, and thoracic empyema (see Table 5). The risk factors for postoperative pneumonia are reported to be smoking habits, high performance status, longer operative procedures, and thoracic or upper abdominal site of operation [25]. We advocate routine preoperative and postoperative physical therapy for elderly patients, particularly those who smoke.

It is important that preoperative cardiopulmonary function be carefully evaluated in patients undergoing operation for lung cancer [26]. Our criteria for operability and pulmonary resection are the following: predicted postoperative VC of 0.8 L/m^2 or greater and predicted postoperative FEV$_1$ of 0.6 L/m^2 or greater. If predicted VC and FEV$_1$, are not adequate to preserve pulmonary function after lobectomy or pneumonectomy, a limited operation such as segmentectomy or wedge resection is an acceptable procedure [27].

In conclusion, the survival rates were equal for two age groups of patients treated surgically for lung cancer, a group 70 years or older and a younger group. The good results were attributed to careful preoperative evaluation, selection of the appropriate surgical procedure, and preoperative and postoperative physical therapy for all elderly patients.

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Ultrasonic Debridement During Mitral Valve Reconstruction for Calcified Mitral Stenosis

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An ultrasonic device in conjunction with open mitral commissurotomy was applied in 8 patients with heavily calcified, stenosed mitral valves. In 6 patients, reconstruction of the mitral valve by debridement of the leaflet calcification with the device was successful. Two patients required valve replacement because of an increase in preexistent mitral regurgitation caused by excessive decalcification. The ultrasonic device proved to be a useful and effective adjunct for salvaging the heavily calcified mitral valve, which would otherwise have to be replaced.

(Ann Thorac Surg 1990;50:923–6)

The heavily calcified mitral valve is not infrequent in elderly patients with mitral stenosis. In most cases, valve replacement is indicated. However, we attempted to debride calcification that extended into the mitral leaflets by means of an ultrasonic tool in conjunction with mitral valve reconstruction. In this paper, we report on the effectiveness and usefulness of ultrasonic decalcification to salvage the stenosed mitral valve.

Material and Methods

Between September 1988 and December 1989, 35 patients underwent open mitral commissurotomy for mitral stenosis, including 11 patients with calcified mitral valves. Ultrasonic debridement in conjunction with open mitral commissurotomy was applied in 8 patients with heavily calcified mitral stenosis. The remaining 3 patients had mild to moderate calcification. Ages ranged from 53 to 63 years old (3 women, 5 men). Preoperative catheterization and echocardiography were carried out in all patients and revealed calcification of the mitral leaflets. All of the patients had severe subvalvar changes with calcification extending into anterior or posterior thickened leaflets. The effective mitral valve area calculated by Gorlin's formula averaged 1.1 cm^2. The diastolic pressure gradient across the mitral valve averaged 8.6 mm Hg. Six patients had concomitant trivial to mild mitral regurgitation preoperatively (Table 1).

The operations were performed with cardiopulmonary bypass, moderate hypothermia, and cardioplegic arrest. Commissurotomy was accomplished with incision of the fused commissures through their length as widely as possible. Complete relief of subvalvar fusions was achieved aggressively by separating the fused and shortened chordae and the papillary muscles by means of sharp dissection. Calcium deposits were debrided meticulously from the leaflets by using an ultrasonic tool provided with irrigation and suction (Cavitron Ultrasonic Surgical Aspirator, Cooper LaserSonic, Inc) (Fig 1). The amplitude of tip vibration and the amount of suction were adjusted to 50% to 60% of maximum strengths. After the procedures, the diameter of the mitral valve orifice was measured with a sizer to confirm that the diameter of the mitral valve orifice was larger than 23 to 25 mm. Competence of the mitral valve was confirmed by intraventricular injection of saline solution under pressure with a hand-held syringe or by digital distortion of the aortic root to create aortic regurgitation. Concomitant procedures were tricuspid annuloplasty in 2 patients and coronary artery bypass grafting in 1. Postoperative valve function was examined by intraoperative pressure measurement and echocardiography after hemodynamics became stable following the termination of cardiopulmonary bypass.

Results

Table 2 summarizes the operative results in 8 patients undergoing ultrasonic debridement in conjunction with open mitral commissurotomy. Six patients had satisfactory results. In these patients, the diastolic pressure gradient across the mitral valve decreased from 8.8 mm Hg (average) to 2.8 mm Hg after operation. Good coaptation with improved mobility of the leaflets and no or trivial mitral regurgitation was confirmed by intraoperative echocardiography and regurgitation testing. All 6 patients have been doing well postoperatively and are in class I according to the New York Heart Association functional classification. Mitral valve areas ranged from 1.5 cm^2 to 3.1 cm^2 with an average of 2.1 cm^2, measured by echocardiography 3 months (average) after operation.

In the remaining 2 patients, valve reconstruction was unsatisfactory and valve replacement was required because preexistent regurgitation was increased owing to poor coaptation of the leaflets, the cause of which was excessive decalcification.

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Table 1. Preoperative Findings of Mitral Stenosis in 8 Patients Undergoing Ultrasonic Debridement in Conjunction With Open Mitral Commissurotomy for Calcified Mitral Stenosis

			Pathological Findings of Mitral Valve			Preop Catheterization		UCG
Patient No.	Age	Sex	Calcification	Subvalvar Changes	Regurgitation	Diastolic Pressure Gradient (mm Hg)	MVA (cm^2)	MVA (cm^2)
1	56	F	Severe	Severe	Mild (1/4)	11	0.6	0.7
2	55	M	Severe	Severe	Mild (1/4)	7	1.0	0.9
3	54	M	Severe	Severe	Moderate (2/4)	9	1.9	1.6
4	53	M	Severe	Severe	Mild (1/4)	5	1.4	1.5
5	63	F	Severe	Severe	None	12	0.7	0.56
6	43	M	Severe	Severe	Mild (1/4)	4	1.0	0.9
7	55	M	Severe	Severe	None	. . .	1.4	1.4
8	60	F	Severe	Severe	Mild (1/4)	12	0.7	0.9
Mean ± SD						8.6 ± 3.3	1.1 ± 0.4	1.05 ± 0.4

MVA = mitral valve area; SD = standard deviation; UCG = ultrasonic cardiography.

Comment

Our policy has been to salvage the stenosed mitral valve whenever possible because of reconfirmation of the limitations of existing prosthetic valves. In 1987, we [1] reported that open mitral commissurotomy provided excellent long-term (14-year follow-up) results in 364 patients with mitral stenosis, even when associated with severe subvalvar changes with or without mild to moderate valve calcification. We [2] also demonstrated that there was no evidence of any adverse effects of mild to moderate calcification on postoperative valve function, either at rest or during exercise, and no influence on the reoperation-free survival rate. However, if the leaflets are heavily calcified, valve replacement is usually recommended.

Recently, Eguaras and colleagues [3] reported an experimental study on surgically excised mitral and aortic calcified valves and confirmed that ultrasonic energy was effective in disintegrating the calcium without damaging the normal valve tissue. At present, the ultrasonic device has been utilized in other cardiac surgical procedures. Vander Salm [4] reported a new technique for mitral valve replacement in patients with mitral annular calcification by complete ultrasonic decalcification. Johnson and colleagues [5] applied the ultrasonic device to debride calcified pericardium in constrictive pericarditis. This device has also been used for the debridement of calcified aortic valve. We report here ultrasonic debridement of calcified mitral valves in conjunction with open mitral commissurotomy. The ultrasonic device appeared to be a useful and effective adjunct to obtain more favorable mobility of the leaflets. The calcific deposits were easily disintegrated and quickly aspirated by the ultrasonic aspirator. However,

Table 2. Operative Results in 8 Patients Undergoing Ultrasonic Debridement in Conjunction With Open Mitral Commissurotomy for Calcified Mitral Stenosis

	Diastolic Pressure Gradient (mm Hg) During OMC		Postop UCG		
Patient No.	Before	After	MVA (cm^2)	Regurgitation	Outcome
1	13	4	1.9	Mild (1/4)	Successful, NYHA I
2	10	2	3.1	None	Successful, NYHA I
3	6	. . .	. . .	. . .	MVR required
4	8	3	2.0	Mild (1/4)	Successful, NYHA I
5	8	. . .	. . .	. . .	MVR required
6	4	2	2.1	None	Successful, NYHA I
7	8	0	2.1	None	Successful, NYHA I
8	10	6	1.5	None	Successful, NYHA I
Mean ± SD	8.8 ± 2.7	2.8 ± 1.8	2.1 ± 0.5		

MVA = mitral valve area; MVR = mitral valve replacement; NYHA = New York Heart Association functional class; OMC = open mitral commissurotomy; UCG = ultrasonic cardiography.

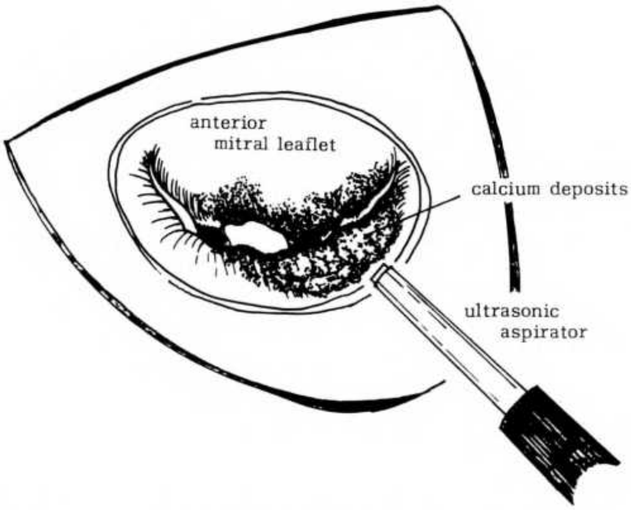

Fig 1. Ultrasonic decalcification employing an ultrasonic aspirator after open mitral commissurotomy for calcified mitral stenosis.

excess decalcification at the rough zone of the leaflets may lead to their poor coaptation due to valve tissue defects. In 2 of our patients, valve replacement was required because of an increase in preexistent regurgitation after decalcification.

We recommend ultrasonic debridement of leaflet calcification to salvage the heavily calcified stenosed mitral valve. Although our experiences are limited and long-term follow-up is needed, we believe that further application of ultrasonic debridement in conjunction with open mitral commissurotomy is warranted.

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INVITED COMMENTARY

With this article, Dr Nakano and colleagues have suggested another use for the surgical ultrasonic aspiration device. Although their report appears promising, my colleagues and I would offer a word of caution and mention our current application of the device.

Until now the published use of the surgical ultrasonic aspiration device in valve operations has been limited to the aortic valve. The aortic root is easily accessed by the existing probe, but the mitral annulus has been more of a challenge. The posterior leaflet and annulus can be routinely debrided, but the anterior-septal area is very difficult to reach through the standard left atriotomy. Alternative incisions, through the atrial septum or through the roof of the atrium, may be quite helpful.

The combined experience of many cardiac surgeons using this device for debridement of stenotic aortic valves has been frustratingly poor. These results have been published and discussed at recent meetings of the Southern Thoracic Surgical Association, by Dr Craver [1], and of the Western Thoracic Surgical Association, by Dr McBride [2]. Although our experience with this device for relief of aortic stenosis has been limited, we too have been disillusioned by restenosis and progression of insufficiency. Similar results, over a longer period of follow-up, might be anticipated with the application of this technology to reconstruction of calcified mitral valves. It may be that ultrasonic debridement damages the underlying valve leaflet in some manner. The tip of the probe does become extremely heated and there are insufficient data regarding detailed microscopic evaluations of tissue that has been decalcified ultrasonically.

It is, however, well documented that restenosis and progressive insufficiency may occur with some regularity after non-ultrasonic, mechanical valve debridement. These data in combination with the more recent ultrasonic aspiration device experience would suggest that stenotic, heavily calcified leaflets may be structurally and functionally diseased beyond the obvious distortion and rigidity that is amenable to macroscopic debridement. Removal of enough calcium to relieve the stenosis (and to preclude "restenosis") may simply leave a less rigid, still abnormal leaflet to inexorably become insufficient. The problems associated with prosthetic valves require that we continue to look at this and other options for valve salvage, but at the present, when very limited debridement of the heavily calcified valve will not permit valve reconstruction, valve replacement is probably the best available option.

Although some may conclude from this that the surgical ultrasonic aspiration device may have no role in the cardiac surgeon's armamentarium, we would suggest that it is more than "a device looking for an operation." Although the individual software expenses are not negligible and must be considered, we have been very pleased with the occasional use of the surgical ultrasonic aspiration device in a variety of situations. We have previously reported the benefits of its use in the debridement of calcified, constrictive pericardium. We have employed it occasionally to thoroughly and safely debride a heavily calcified annulus in preparation for valve replacement. Occasionally, the heavily calcified aortic root represents a challenge to closure and the device can quite safely decalcify or endarterectomize the involved area without creating flaps. Finally, at reoperation for coronary revascularization, dense vascular adhesions may be encountered. In such instances the device can be used to dissect bloodlessly, leaving previous grafts and pledgets undamaged.

The technique for the use of the device is simple, but it does have a learning curve. Depending on the orientation of the tip and the setting of the device's amplitude and suction it can dissect through bone, or it may incise a myocardial muscle bridge while sparing the coronary artery underneath. It should be noted that the suction serves not only to remove the small bits of debris generated, but also to attract soft tissue to the energy source. For this reason increasing the suction may increase the risk of injuring adjacent soft tissue at any given amplitude setting. The technique of dissection of adhesions involves using the device tip so it is directed perpendicular to the tissue to be rendered, but tangential to the tissue to be spared. A sweeping motion is employed, and if set appropriately (start at approximately 50% of full amplitude and 50% full suction) the device still leaves the toughest avascular, fibrous strands behind. If the tip is brought to bear on these strands they will be disrupted, but it is faster if the assistant has scissors ready to lyse the isolated adhesions to speed the process along as the surgeon dissects out a broad area. The irrigation pedal on the device, or other irrigation from the field, should be used occasionally to keep the heat of the tip localized.

We recognize the contribution that Dr Nakano and associates have made in describing the use of the surgical ultrasonic aspiration device for mitral valve salvage. Their experience will provide a cohort for follow-up so we can determine whether the long-term results will be similar to those of aortic debridement. In the meantime we will continue to selectively apply the device in a variety of other cardiac operations.

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Repair of Flail Anterior Leaflets of Tricuspid and Mitral Valves by Cusp Remodeling

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We present an alternative approach to extensive rupture of the chordae tendineae leading to flail anterior leaflets. Resection of the affected cusp segment, suture of the cut edges, and extensive plication of the segment of annulus devoid of leaflets abolished massive regurgitation while maintaining an adequate valve orifice.

(Ann Thorac Surg 1990;50:927–30)

Rupture of chordae tendineae can follow a variety of pathological conditions. We present an alternative approach to extensive chordal rupture after tricuspid trauma and after mitral valve bacterial endocarditis.

Material and Methods

Patient 1

A 30-year-old man had noted repeated bouts of leg edema and dyspnea of 3 years' duration. His exercise tolerance had deteriorated progressively and allowed walking up only ten stairs. His history revealed that during childhood, he had fallen from a first floor window and sustained minor external thoracic trauma.

On admission to our service, the patient was free from dyspnea and cyanosis, and the jugular veins were not prominent. A grade 3/6 systolic murmur was present over the left sternal border in the fifth intercostal space. Chest roentgenograms showed massive enlargement of both the right atrium and the right ventricle. Two-dimensional echocardiography confirmed these findings and also demonstrated severe tricuspid insufficiency. The left ventricle, aorta, and pulmonary arteries were normal. At right heart catheterization in an outside hospital, normal pressures were found. The pulmonary artery oxygen saturation was markedly reduced on exercise (37% at 50 W), and massive tricuspid regurgitation was again noted.

Surgical intervention was undertaken on February 2, 1989, using total cardiopulmonary bypass and cold potassium cardioplegia. The tricuspid annulus was enlarged to such an extent that a woman's fist could easily have passed through it. The caudad two thirds of the chordae of the anterior leaflet was torn. Likewise, the chordae tendineae of the posterior leaflet were ruptured except for those supporting it at its commissure with the septal leaflet, which appeared intact (Fig 1). The flail portions of the anterior and posterior leaflets were resected (Fig 2A). Next, the cut edges of the leaflets were united by a running 5-0 suture. Finally, the segment of the annulus devoid of leaflets after resection was plicated by 3-0 mattress sutures supported with pericardial pledgets (Fig 2B). The resulting tricuspid valve orifice measured 3 cm^2.

A catheter was passed into the right ventricle through the distally clamped pulmonary artery, and the right ventricle was inflated with cardioplegic solution. The valve appeared fully competent (Fig 3).

Two-dimensional echocardiography performed 10 days postoperatively showed grade I residual tricuspid insufficiency. The patient is doing well and has regained full exercise tolerance.

Patient 2

A 21-year-old man with a negative history with respect to valve disease experienced intermittent fever, abdominal symptoms, and polyarthritic pain 8 months before admission to our service. Seven months before referral, a grade 4/6 mitral diastolic murmur was discovered, and he was hospitalized elsewhere for 3 months. Echocardiography revealed massive mitral insufficiency with vegetations on the valve. The heart was markedly enlarged. Cardiac catheterization demonstrated a pulmonary artery pressure of 40/14 mm Hg and a mean pulmonary capillary pressure of 18 mm Hg. After a prolonged course of antibiotic treatment, the patient was discharged from that hospital with mitral insufficiency grade III to IV and was scheduled for an open heart operation.

At operation on September 2, 1988, the mitral valve was approached through the roof of the left atrium. The valve leaflets and suspension apparatus appeared unremarkable except for rupture of all chordae of the anterior leaflet from the posterior strut chorda, which was intact, to the posterior commissure (Fig 4). As a result, one quarter of the surface of the anterior leaflet was prolapsing. The corresponding segment of the annulus was dilated. There were minor vegetations on the ruptured chordae.

The entire flail part of the anterior leaflet from the posterior strut chorda to the posterior commissure was resected. Next, beginning at the posterior commissure, the posterior leaflet was detached from the mitral annulus for a distance equal to that of the cut edge of the remaining anterior leaflet. The commissural tip of the mobilized portion of the posterior leaflet was inverted to meet the anterior leaflet at the point of insertion of the strut chorda

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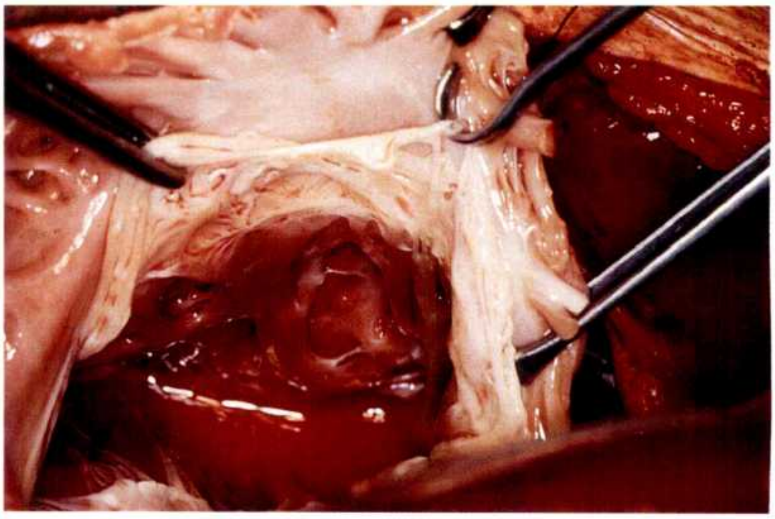

Fig 1. Incompetent tricuspid valve. Note that large portions of the anterior and posterior leaflets are flail as a result of chordal rupture.

(Fig 5). The cut edges of both leaflets were united using a continuous 4-0 monofilament suture. In this way, the chordae of the detached segment of the mural leaflet were rotated and at the same time were transposed anteriorly, thereby making them support the new commissure thus created (Fig 6). The annulus segment devoid of leaflets was obliterated by extensive plication using deeply biting 3-0 mattress sutures supported by pericardial pledgets. The opening surface of the valve after reconstruction measured 3.2 cm^2. Intraoperative testing with cardioplegic solution infused into the left ventricle with a catheter inserted through the aorta revealed a fully competent valve.

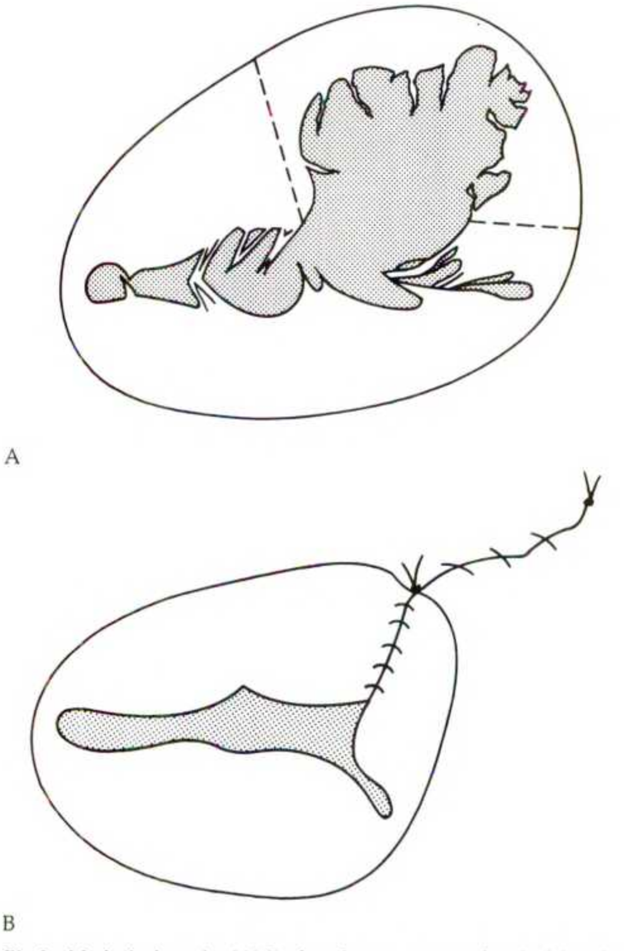

Fig 2. Method of repair. (A) Broken lines *correspond to incisions in leaflets. (B) The cut edges of the remaining cusps are sutured. The portion of the annulus devoid of leaflets after resection is extensively plicated, with the plication carried out onto the atrial wall.*

The postoperative course was uneventful. Two-dimensional echocardiography at discharge proved the absence of mitral insufficiency. The patient is doing well and is fully active.

Comment

Since its description by Williams [1] in 1829, tricuspid insufficiency has been a rare complication of nonpenetrating chest trauma [2–21]. To date, 31 cases of traumatic tricuspid insufficiency have been reported in the literature [1–21]. All patients except 2 were male, and the mean age was 36 years (range, 10 to 70 years). The main cause of traumatic tricuspid insufficiency was a car accident (77% of patients).

Depending on the extent of damage to the valve-supporting structures and leaflets, the initial clinical course of patients with traumatic tricuspid insufficiency can be benign [9]. The interval from injury to surgical repair has ranged from 2 weeks to 32 years (mean interval, 10 years) [2, 9, 16]. Preponderant findings at operation included ruptured chordae or ruptured papillary muscles or both in approximately 50% of patients. The anterior subvalvar portion was involved in two thirds of all patients. Isolated leaflet tears were less frequent, occurring in 36%. Again, there was a preponderant involvement of the anterior leaflet in three fourths of the latter patients.

The most common method of repairing a tricuspid valve with torn chordae has been bicuspidization based on the technique of Kay and colleagues [22, 23]. In our patient with excessive rupture of chordae tendineae of two leaflets, a Kay valvoplasty either would have been

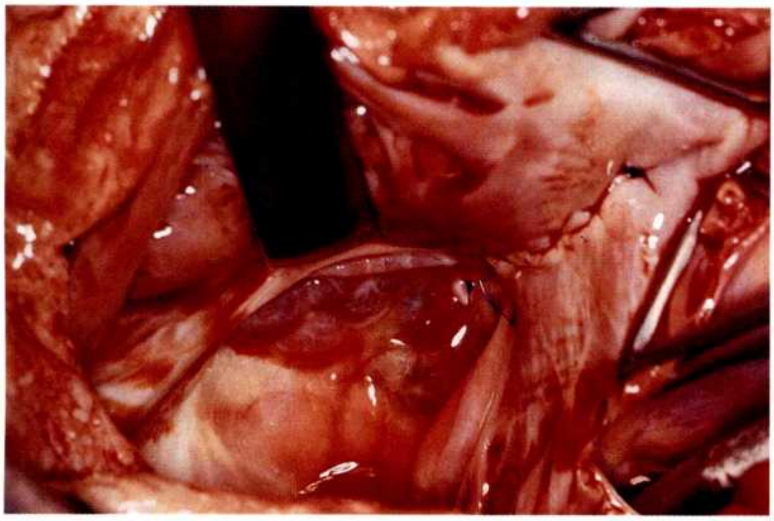

Fig 3. Reconstructed valve. It is competent when the right ventricle is filled with saline solution under pressure.

ineffective or would have resulted in a critically narrow orifice. We therefore selected a method that would preserve as much of the free leaflet substance as possible: leaflet resection, suture of the cut edges of the anterior and posterior cusps, and additional annuloplasty. In this way, the valve retained proper function without stenosis.

Rupture of chordae tendineae of the anterior mitral leaflet is a common finding after acute bacterial endocarditis and usually leads to valve replacement. However, limited resection of the anterior mitral cusp has been described by McGoon [24], and Carpentier and associates [25] have shown the feasibility of transposing a segment of the posterior cusp for filling a defect in the free anterior leaflet, thereby supporting its margin. Neither of these methods has been used to repair an anterior mitral leaflet devoid of its commissural support, and neither seems applicable for this condition. In our patient, all chordae caudad to the posterior strut chorda were torn. We therefore decided to create a new commissure by rotating a segment of the posterior leaflet, detached from the annulus, around its supporting point. This resulted in a fully competent valve of adequate size.

Function of flail anterior leaflets of both the tricuspid and mitral valves can be restored by resection of the affected cusp segment, suture of the cut edges, and plication of the annulus devoid of leaflets. The procedures described provide an adequate valve orifice by maximal use of the remaining leaflets and their chordal support.

Addendum

Since the submission of this manuscript, we have operated on 2 more patients demonstrating the type of pathological findings described. A 22-year-old woman was seen with grade III to IV mitral insufficiency, supposedly a result of bacterial endocarditis at age 11 years. At operation, all chordae tendineae anterior to the anterior strut chorda were torn. Reconstruction followed the method shown in Figures 1 through 3, though on the opposite side of the valve. Four weeks after operation, echocardiography

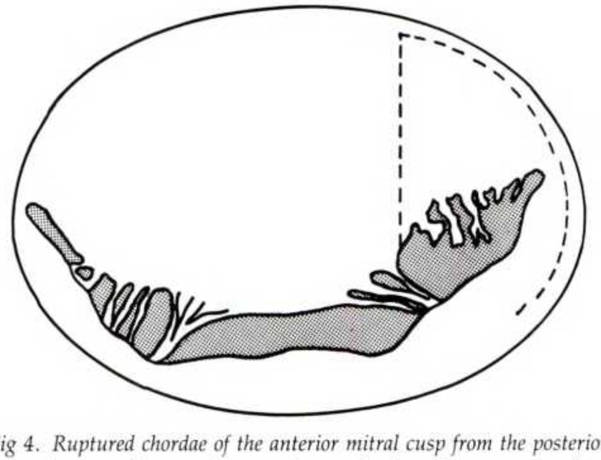

Fig 4. Ruptured chordae of the anterior mitral cusp from the posterior strut chorda to the posterior commissure. Broken lines *correspond to incisions in leaflets.*

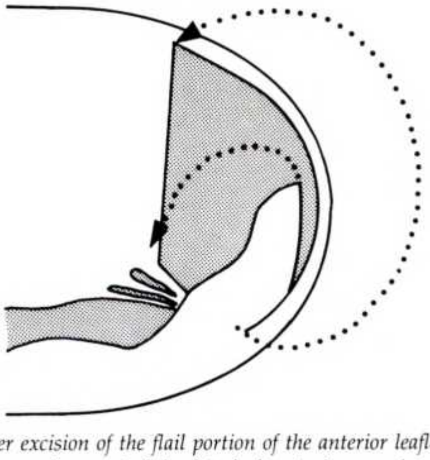

Fig 5. After excision of the flail portion of the anterior leaflet, the tip of the corresponding part of the detached posterior cusp is rotated toward the torn edge of the former.

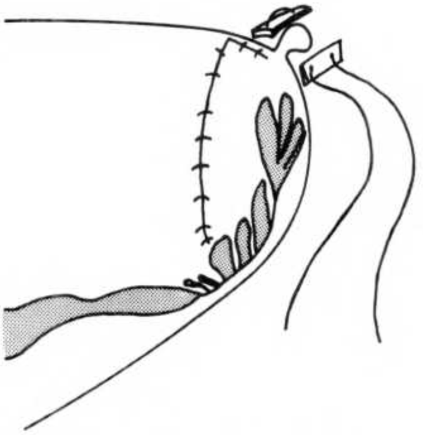

Fig 6. Anterior and posterior leaflets are sutured, thereby creating a neocomissure.

revealed substantial reduction in cardiac size and only trivial mitral insufficiency.

The second patient, a 19-year-old man, was kicked in the sternum by a horse when he was 15 years old. The findings were excessive cardiomegaly and right ventricular failure caused by massive tricuspid insufficiency. At operation, he showed exactly the same pathology as depicted in Figures 4 through 6 except that there were three 2- to 3-mm perforations in the hinge line of the flail anterior leaflet (which were sutured). The valve was reconstructed in an identical manner, with the addition of a De Vega valvoplasty to reinforce the annulus. On echocardiography 2 weeks after operation, there was no regurgitation through the reconstructed valve.

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Thoracic Spinal Fractures and Aortic Rupture: A Significant and Fatal Association

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We analyzed the frequency of occurrence of traumatic aortic rupture (TAR) in patients with and without thoracic spinal fractures. Among 4,676 blunt chest trauma victims admitted to the hospital between 1972 and 1988, 148 (3.2%) suffered one or more thoracic vertebral fractures. There were 73 patients with one or more fractures of the first eight thoracic vertebrae (T1 to T8); of these 73, 4 also suffered TAR (5.5%). There were 4,603 patients without fractures of T1 to T8, and 64 of these patients also suffered TAR (1.4%). This difference was significant by the χ^2 and Fisher exact tests, $p = 0.00378$ and $p = 0.021003$, respectively. Additionally, all 5 patients with TAR and thoracic vertebral fractures died. We conclude that patients with one or more fractures of T1 to T8 have a statistically significant increase in the incidence of TAR.

(Ann Thorac Surg 1990;50:931–3)

The thoracic spine provides a mechanical base of support for the chest and its contents because it is a rigid structure between the relatively mobile cervical and lumbar spines. It is capable of flexion, extension, and rotation, but is considerably less mobile than other parts of the spine. The thoracic spine owes its relative rigidity to its internal structure, and to the supporting ribs and sternum [1]. It is rarely the site of minor strains like those of the more mobile cervical and lumbar parts of the spine. The stability of the dorsal spine is illustrated by the rarity of thoracic disc herniations. The rigidity of the thoracic spine limits injury to it. Conversely, injury to the thoracic spine implies that a large amount of force has been sustained [1, 2]. Consequently, patients who have sustained thoracic vertebral fractures might be anticipated to have incurred other severe injuries [1]

The descending thoracic aorta lies in close proximity to the thoracic vertebrae, and begins at the level of the fourth thoracic vertebra [3]. The most common site of thoracic aortic rupture (TAR) is just distal to the origin of the left subclavian artery at the level of the sixth thoracic vertebra. Accordingly, patients who have suffered fractures to the upper thoracic vertebrae have sustained a great amount of force to the area of the chest where TAR most frequently occurs.

It has been suggested that fractures of the dorsal spine may be associated with TAR [1], but this assumption has not been analyzed statistically in a large group of patients with blunt chest trauma. This study tested the null hypothesis that TAR does not occur more commonly in patients with thoracic vertebral fractures.

Material and Methods

The St. Paul-Ramsey Trauma Registry has catalogued 4,676 patients with blunt chest trauma between the years 1972 and 1988. These 4,676 patients comprise the basis of this report. There were 148 patients of the 4,676 (3.2%) who suffered one or more fractures of the thoracic vertebrae; 115 were male and 33 female. The patients ranged in age from 9 to 80 years. The majority were injured in motor vehicle accidents.

Two statistical calculations were carried out to determine the association between fracture of thoracic vertebrae and TAR. First, the frequency of TAR among patients with one or more fractures of any thoracic vertebrae was compared with the frequency of TAR in patients with no fractures of thoracic vertebrae. Second, the frequency of TAR in patients with fractures of T1 to T8 was compared with the frequency of TAR in patients without T1 to T8 fractures. Statistical analysis was carried out using the χ^2 and Fisher exact tests. The Fisher exact test was used when any one of the cells of the contingency table was 5 or less.

Results

There were 5 of 148 patients with thoracic vertebral fractures who also suffered TAR (3.4%). There were 63 of 4,528 patients with TAR and no thoracic vertebral fractures (1.4%). Although this difference was significant by χ^2 analysis ($p = 0.04659$), Fisher's exact test was used because one cell contained 5 patients. By Fisher's exact test, $p = 0.0629$. Statistical analysis was then carried out for patients with vertebral fractures involving T1 to T8. Four patients with one or more fractures of T1 to T8 suffered TAR (5.5%). Sixty-four of 4,603 patients with no fracture of T1 to T8 suffered aortic rupture (1.4%). The χ^2 test showed $p = 0.00378$ and Fisher's exact test showed $p = 0.021003$.

All five patients with TAR and thoracic spine fractures

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suffered profoundly severe injuries. Only 1 of these 5 patients survived to undergo thoracotomy. This 69-year-old male victim of a motor vehicle accident arrived at the hospital with a blood pressure of 70 mm Hg. Results of peritoneal lavage were positive, and the patient underwent laparotomy for a ruptured liver and diaphragm. A widened mediastinum was noted on chest roentgenogram and arch aortography was carried out. This showed TAR and a left thoracotomy was performed. Left atriofemoral bypass was performed, but cardiac arrest ensued before the repair could be done. Resuscitation was unsuccessful.

The second of these patients was a 71-year-old female pedestrian struck by a car. The patient was seen at the emergency department with a blood pressure of 60 mm Hg. Results of peritoneal lavage were positive and celiotomy was performed. Massive liver injuries were found, and the patient died intraoperatively owing to hypovolemia and multiple injuries. Resuscitation failed and TAR was found at postmortem examination.

Three other patients were seen at the hospital with no blood pressure and were receiving cardiopulmonary resuscitation. One was a 48-year-old male pedestrian, another a 23-year-old male victim of a motor vehicle accident, and the last was a 50-year-old man crushed between a forklift and a wall. None of these 3 could be resuscitated and TAR was found at autopsy along with other massive injuries.

Comment

Blunt rupture of the thoracic aorta implies that the victim has incurred a severe amount of force. As a consequence, severe associated injuries may be anticipated with patients who suffer TAR. Many authors have attempted to correlate certain injuries with the occurrence of TAR. Multiple rib fractures, flail chest, fractured first or second rib, fractured sternum, massive hemothorax, and thoracic vertebral fractures have all been implicated as harbingers of TAR [4–13]. The association of these injuries with TAR has not been demonstrated conclusively by statistical analysis.

Fractures of the first rib have been implicated as indicating the presence of severe thoracic and extrathoracic trauma. Richardson and associates [7] studied 55 patients with first rib fractures. Associated chest injuries were found in 35 of these 55 patients, and concomitant abdominal visceral trauma was seen in 18. The chest injuries included hemopneumothorax, pulmonary contusion, and flail chest. The abdominal injuries consisted of ruptured spleen, liver laceration, and pancreatic injury. Head injuries were found in 29 patients. Three patients (5.5%) sustained subclavian artery injuries. Importantly, there were no instances of aortic rupture.

Phillips and co-workers [8] studied 45 patients with first rib fractures. Four of these patients sustained subclavian artery injuries (8.8%). None of the victims suffered TAR.

The tremendous force necessary to cause a fractured sternum has led some clinicians to suggest that the presence of sternal fracture predicts the association of severe associated injuries, in particular TAR [4, 9, 10]. The association of sternal fractures with TAR has been conclusively analyzed statistically in only one investigation [14]. The authors of this study showed that there was no statistically significant increase of TAR in patients with sternal fractures. Previous authors have claimed there was an association between sternal fracture and TAR, but their cases were not analyzed statistically [13, 15].

A displaced first rib fracture, the association of a first rib fracture with a fracture of the clavicle or scapula, a fractured scapula, and the combination of a clavicle and scapula fracture on the same side have been suggested as portending aortic rupture [5]. These correlations have not been proved.

We showed that there was a significant increase of TAR in patients with fractures of T1 to T8. The area surrounding T1 to T8 is a contiguous region above and below the site where the thoracic aorta most commonly ruptures. As a result, aortic rupture would be expected to occur more commonly in patients with thoracic spinal fractures. However, the 5 patients with TAR and thoracic vertebral fractures who we have reported here had been severely injured. Only 1 of these 5 survived long enough to undergo thoracotomy. This 1 patient represents only 1 of 37 (3%) previously reported TAR patients treated surgically [9].

The diagnosis of aortic rupture is usually made by the interpretation of a widened mediastinal shadow on chest roentgenogram [16]. A variety of other clinical clues suggest that TAR may be present [16–18]. The present study indicates that patients who suffer thoracic vertebral fractures should be considered to potentially have suffered aortic rupture, and may need thoracic aortography.

We conclude that TAR occurs significantly more often with thoracic spinal fractures, and that the simultaneous occurrence of these injuries is highly lethal.

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Circulatory Support 1991

The Second International Conference on Circulatory Support Devices in End-Stage Heart Disease

San Francisco Marriott Hotel, San Francisco, California
November 15–17, 1991 (weekend meeting)

Circulatory Support 1991 will be a comprehensive multidisciplinary meeting focusing on the clinical applications of the entire spectrum of circulatory support devices. Surgeons, cardiologists, anesthesiologists, intensivists, perfusionists, engineers, and operating room and intensive care nurses are encouraged to attend as individuals or as a team.

The meeting is sponsored by The Society of Thoracic Surgeons under the direction of its Committee on Circulatory Assist Devices and Artificial Hearts. Committee members are: D. Glenn Pennington, MD, Chairman; Jack G. Copeland, MD; Charles Hahn, MD; J. Donald Hill, MD; George J. Magovern, MD; Peer M. Portner, PhD; and Glenn Rahmoeller.

For this weekend meeting, the Saturday morning segment entitled "Problem Cases" will include panel and audience discussions of four major subject areas: patient selection; anticoagulation management, bleeding, and thromboembolic risk; infections—prophylaxes and treatment; and management of secondary organ dysfunction. An outstanding international panel has been chosen to represent a broad spectrum of opinions for each subject.

Late Saturday morning and early afternoon, video sessions on the working details and proposed indications for support devices that are currently in use or development will be presented. A report of the current circulatory support device registry will be given. Breakout sessions of smaller groups will follow and focus on how to use external nonpulsatile circulatory support, external pulsatile circulatory support, and internal pulsatile circulatory support.

The Sunday sessions from 7:45 AM to 1:00 PM will be devoted to state-of-the-art presentations covering: emergency resuscitation techniques, heparinless bypass, the Hemopump experience, postoperative use of the Biopump, the VAD experience in Japan, optimizing hemodynamics and assessing myocardial recovery, right heart management during LVAD support, circulatory support in children, bridge to transplantation with the Thoratec VAD and with the Symbion TAH, chronic support with the TCI left ventricular assist system, the Novacor left ventricular assist system, and the Penn State TAH.

Poster presentations concerning a wide variety of subjects related to circulatory support as well as commercial exhibits demonstrating currently available devices will be available during breaks. Full CME credit will be provided for attendees.

For registration details concerning poster submissions and other available meeting information contact: The Society of Thoracic Surgeons, 401 N Michigan Ave, Chicago, IL 60611; telephone: (312) 644-6610.

Cardioplegia-Induced Damage to Ischemic Immature Myocardium Is Independent of Oxygen Availability

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The known benefits of hypothermic pharmacological cardioplegia in protecting the ischemic adult heart may not extend to children. Protection of the ischemic immature rabbit heart with hypothermic Krebs-Henseleit bicarbonate buffer is better than with hypothermic St. Thomas' II cardioplegic solution. We investigated whether the availability of oxygen in the preischemic perfusate is responsible for the increased tolerance to ischemia of immature (7- to 10-day-old) hearts perfused with Krebs buffer in comparison with St. Thomas' II solution immediately before ischemia. After obtaining preischemic control data in the "working" mode, we perfused hearts (n = 8 per group) for 3 minutes with hypothermic (14°C) Krebs buffer or hypothermic St. Thomas' II solution saturated with 0%, 25%, or 95% oxygen. This was followed by 2 hours of global ischemia at 14°C. Hearts were reperfused for 15 minutes in the Langendorff mode and 35 minutes in the working mode, and recovery of function was measured. For preischemic oxygen concentrations of 0%, 25%, and 95%, recovery of aortic flow in hearts protected by hypothermia alone during ischemia was 74% ± 9%, 82% ± 4%, and 99% ± 2% of preischemic values, respectively. In hearts protected by hypothermia plus cardioplegia, the values were 69% ± 6%, 72% ± 3%, and 86% ± 5%, respectively. Thus, at equal oxygen concentrations, recovery of postischemic function was better in hearts protected by hypothermia alone compared with hypothermia plus cardioplegia. We conclude that factors other than oxygen availability are responsible for the damaging effect of St. Thomas' II solution on the ischemic immature rabbit heart.

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Hypothermia combined with pharmacological cardioplegia protects the globally ischemic adult heart [1], but this benefit may not extend to children [2]. We [3] have shown that protection of the ischemic immature rabbit myocardium with hypothermic Krebs-Henseleit solution is better than with hypothermic St. Thomas' II cardioplegic solution. This effect is both age dependent [3] and species dependent [4]. The two preischemic perfusates used in our studies, by nature of their composition, have a high and a moderate oxygen saturation: 95% oxygen in Krebs-Henseleit solution and 25% oxygen in St. Thomas' II solution.

Because available oxygen is the dominant factor in the onset and development of myocardial ischemia, we assessed the influence of extracellular oxygen availability on susceptibility to ischemia modified by hypothermia alone and by hypothermia plus cardioplegia in isolated immature rabbit hearts. We wished to determine in this model whether the increased oxygen availability in Krebs buffer during preischemic coronary perfusion was responsible for the superior protection of the ischemic immature rabbit heart with hypothermia alone compared with the decreased protection with the lower oxygen level in hypothermic St. Thomas' II solution.

Material and Methods

Immature New Zealand white rabbits (7 to 10 days old) were obtained from a *Pasteurella*-free breeding colony at the Medical College of Wisconsin. Between 6 and 11 kits were produced per litter. Data on body weight and heart weight are shown in Table 1. Heparin sodium (150 IU/kg) was administered intraperitoneally before anesthesia, which was induced and maintained with halothane (4.0% and 1.5%, respectively). The chest was opened, and the pericardium was removed. The aorta and inferior vena cava were isolated, and the heart was rapidly excised and placed in ice-cold Krebs-Henseleit bicarbonate buffer. The time from opening of the chest to excision of the heart was less than 1 minute.

Perfusion System

We [3] modified the standard isolated "working" heart preparation (Biomedix, Elm Grove, WI), described in 1967 by Neely and colleagues [5], to provide a physiological work load and temperature suitable for immature rabbits. The cannulas for the aorta and left atrium of the immature hearts were 1.8 mm in inner diameter and 2.0 mm in outer diameter, respectively. The outer diameter of the aortic cannula matched the inner diameter of the immature rabbit aorta. The preparation has been described in detail previously [3, 5]. Essentially, this is a left heart preparation in which oxygenated Krebs-Henseleit bicarbonate buffer supplemented with glucose enters the cannulated left atrium at a pressure of 6 mm Hg. The perfusate then

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Table 1. Baseline Measurements[a]

Variable	Preischemic Control Values
Body weight (kg)	0.13 ± 0.01
Heart wet weight (g)	0.7 ± 0.2
Heart rate (beats/min)	294 ± 11
Aortic flow (mL/min)	32 ± 5
Coronary flow (mL/min)	8 ± 1

[a] Data are shown as the mean ± the standard deviation, pooled from all animals used in this study. With the exception of body weight, all data were derived from isolated hearts.

passes to the left ventricle from which it is spontaneously ejected through the cannulated aorta against a compliant hydrostatic head with a mean pressure of 42 mm Hg.

Hearts in which the hemodynamic performance deviated from the mean values shown in Table 1 by one standard deviation or more were excluded from the study. This exclusion criterion resulted in rejection of approximately 15% of the hearts. Additional hearts were perfused until eight were available for analysis of function. The heart and perfusion fluids were kept in temperature-controlled chambers to maintain myocardial temperature at the desired level. During all periods of ischemia, the heart was immersed in ungassed physiological saline solution.

Perfusion Media

The standard perfusion medium was Krebs-Henseleit bicarbonate buffer [6] (pH 7.41 at 39°C and pH 7.25 at 14°C when saturated with 95% oxygen and 5% carbon dioxide) in which the calcium content was reduced to 1.8 mmol/L. To this was added glucose (11.1 mmol/L). The cardioplegic solution was St. Thomas' II solution [1]. Compositions of the individual perfusates are shown in Table 2. Glucose was excluded from all perfusates during hypothermic Langendorff perfusion of the coronary network immediately before and throughout ischemia. Oxygen saturation of the preischemic perfusate was varied, but the pH was maintained by gassing the perfusate with a defined mixture of oxygen, carbon dioxide, and nitrogen. The percentage oxygen saturation was maintained by gassing the perfusates with oxygen at concentrations of 0%, 25%, or 95%; the pH of St. Thomas' II solution and Krebs-Henseleit buffer was maintained at 7.80 and 7.25, respectively, by gassing the perfusates with 0.5% carbon dioxide and 5.0% carbon dioxide, respectively; and the balance of the gas mixture was made up of nitrogen.

During the preparation of all calcium-containing solutions, precautions were taken to prevent the precipitation of calcium by gassing the perfusates with carbon dioxide. Oxygen saturation of the perfusates was measured using a micro-oxygen electrode (Microelectrodes Inc, Londonderry, NH) calibrated against known oxygen standards at 14°C, the temperature of the preischemic coronary perfusates. Oxygen content of the perfusates was measured using a Lex-O_2-Con fuel cell analyzer (Lexington Instruments, Waltham, MA) calibrated against room air at room temperature. All measurements of pH were made using a Data Tech 14 pH meter (Data Tech Systems Ltd, Victoria, BC, Canada) against known pH standards at 14°C. Before use, all perfusion fluids were filtered through cellulose acetate membranes having a pore size of 5.0 μm.

Measurement of Indices of Function

Aortic flow was defined as the ability of the heart to eject perfusate against the hydrostatic head, with flow rate measured using an in-line analog flowmeter (Biomedix). Coronary flow rate was defined by timed collections of the effluent from the right heart into a graduated cylinder. Electrical activity and heart rate were measured using bipolar electrocardiographic leads attached to the aortic and atrial cannulas.

Creatine Kinase Determination

The entire coronary effluent during the Langendorff reperfusion period was collected into a graduated cylinder surrounded by ice. Bovine serum albumin was added to the cylinder to achieve a final concentration of 5% (wt/vol) to stabilize the enzyme. After the volume of effluent was recorded and the contents of the flask were mixed, an aliquot of the coronary effluent was taken for the determination of total creatine kinase (ATP: creatine *N*-phosphotransferase) activity [7].

Perfusion Sequence

OXYGEN AND HYPOTHERMIC PROTECTION. The following experiments were performed in a random manner in three groups of hearts at 0%, 25%, or 95% oxygen saturation of the perfusate to test the null hypothesis that modification of hypothermic ischemia in the immature heart is unaf-

Table 2. Composition of Coronary Perfusates

Component	Krebs-Henseleit Bicarbonate Buffer (mmol/L)	St. Thomas' Hospital Cardioplegic Solution II (mmol/L)
NaCl	118.5	110.0
$NaHCO_3$	25.0	10.0
KCl	4.8	16.0
KH_2PO_4	1.2	. . .
$MgSO_4 \cdot 7H_2O$	1.2	. . .
$MgCl_2 \cdot 6H_2O$	. . .	16.0
$CaCl_2 \cdot 2H_2O$	1.8	1.2
pH[a]	7.41 ± 0.01	7.80 ± 0.01
O_2 saturation (%)[b]	95 ± 2	25 ± 3
O_2 content (mL/100 mL)[c]	2.3 ± 0.1	0.9 ± 0.1

[a] The pH of unmodified Krebs-Henseleit bicarbonate buffer was 7.41 at 39°C and 7.25 at 14°C when gassed with 5% carbon dioxide. The pH of the unmodified cardioplegic solution was 7.80 at 14°C when gassed with 0.5% carbon dioxide. [b] The oxygen saturation of the coronary perfusates at 14°C was determined at 14°C. [c] The oxygen content of the coronary perfusates at 14°C was determined at room termperature. Data on pH, oxygen saturation, and oxygen content represent the mean ± the standard deviation of eight observations.

fected by the availability of extracellular oxygen in the preischemic perfusate. Immediately after aortic cannulation, the hearts were perfused in the Langendorff mode [8] for a 15-minute stabilization period during which the left atrium was cannulated.

The hearts were then converted to the working mode [5] for 35 minutes. During this period, control values for aortic and coronary flow rate, heart rate, and electrical activity were recorded under steady-state conditions. After perfusion in the working mode, hearts were subjected to a single 3-minute period of hypothermic (14°C) coronary perfusion in the Langendorff mode with Krebs-Henseleit bicarbonate buffer saturated with either 0%, 25%, or 95% oxygen. Hearts were then subjected to hypothermic (14°C) global ischemia for 2 hours. This is the target temperature usually achieved clinically for cardioplegic arrest of the heart. The 3-minute period of preischemic coronary perfusion was carried out with perfusion pressure fixed at 60% of the mean aortic pressure for the immature rabbit heart. This reduced pressure was selected to minimize the development of iatrogenic myocardial edema while maintaining adequate volume of perfusion and cooling of the heart.

After the ischemic period, hearts were reperfused for 15 minutes in the Langendorff mode with the entire coronary effluent collected for the determination of creatine kinase activity. Hearts were then converted to the working mode for 35 minutes, during which time the various indices of function were measured under steady-state conditions and expressed as a percentage of the preischemic control values. Each heart thus served as its own control.

OXYGEN AND CARDIOPLEGIC PROTECTION. The following experiments were performed in a random manner in an additional three groups of hearts at 0%, 25%, or 95% oxygen saturation of the perfusate to test the null hypothesis that modification of hypothermic ischemia in the immature heart by St. Thomas' II cardioplegia is unaffected by the availability of extracellular oxygen in the preischemic perfusate. The protocol used for this experiment was the same as that for the previously described study except that St. Thomas' II cardioplegic solution was used to perfuse the heart during the 3-minute period immediately before ischemia.

Expression of Results

Aortic flow rate was expressed as a percentage of its preischemic control value. Creatine kinase leakage was expressed as international units released per 15 minutes per gram dry weight. Eight hearts were used for each condition studied, and the results were expressed as the mean ± the standard deviation of the mean. Statistical analysis was performed using the Kruskal-Wallis nonparametric analysis of variance by ranks as a first step, and where this proved significant, the Mann-Whitney test was used as a second step to identify which groups were significantly different [9]. Significance was accepted at the level of p less than 0.05.

Animals used in this study received humane care in compliance with the "Principles of Laboratory Animal Care" formulated by the National Society for Medical Research and the "Guide for the Care and Use of Laboratory Animals" published by the National Institutes of Health (NIH publication No. 85-23, revised 1985).

Results

Baseline Measurements

Baseline data for the immature rabbit heart are shown in Table 1. The hemodynamic stability of the model was assessed by perfusing six hearts in the working mode for 120 minutes with nonrecirculating perfusate. No significant changes in heart rate or aortic flow from the values reported in Table 1 occurred until after 85 minutes of perfusion in these immature hearts. In the studies described that test our hypotheses, hearts were perfused in the working mode for a maximum of 35 minutes, well within the stability limits of the preparation.

Oxygen and Preischemic Perfusion

The time to diastolic arrest from the onset of hypothermic coronary perfusion immediately before ischemia was dependent on both the oxygen saturation and the ionic composition of the perfusate. In hearts where the perfusate was saturated with 0%, 25%, or 95% oxygen, the time to diastolic arrest on perfusion with cold Krebs buffer was 23 ± 3, 52 ± 16, and 105 ± 26 seconds, respectively, and with St. Thomas' II solution, significantly shorter at 16 ± 2, 18 ± 3, and 20 ± 3 seconds, respectively ($p < 0.05$ Krebs buffer versus St. Thomas' II solution for each oxygen saturation).

The total volume of coronary effluent collected during the 3-minute period of coronary perfusion before ischemia was similar regardless of oxygen content or ionic composition of the perfusate. In hearts where the perfusate was saturated with 0%, 25%, or 95% oxygen, the total volume collected on perfusion with cold Krebs buffer was 34 ± 3, 31 ± 4, and 27 ± 5 mL, respectively, and with St. Thomas' II solution, 32 ± 3, 30 ± 3, and 28 ± 4 mL, respectively (not significant).

Oxygen and Hypothermic Protection (Krebs Buffer)

When the coronary perfusate was saturated with 95% oxygen immediately before ischemia, recovery of aortic flow in hearts protected with hypothermia alone during ischemia was 99% ± 2% (Fig 1). When the oxygen saturation of the preischemic coronary perfusate was reduced to 25%, recovery of aortic flow declined to 82% ± 4% ($p < 0.05$, 95% oxygen versus 25% oxygen). Even when the preischemic perfusate was anoxic (0% oxygen), recovery of aortic flow was 74% ± 9% of the preischemic control value ($p < 0.05$, 95% oxygen versus 0% oxygen). All hearts returned spontaneously to normal rhythm on reperfusion. There were no significant changes in heart rate on reperfusion for any of the preischemic oxygen saturations investigated. Thus recovery of postischemic aortic flow was directly related to the extent of oxygen saturation during preischemic coronary perfusion.

Oxygen and Cardioplegic Protection (St. Thomas' II Solution)

Recovery of postischemic function for hearts protected with hypothermia alone during ischemia is compared in

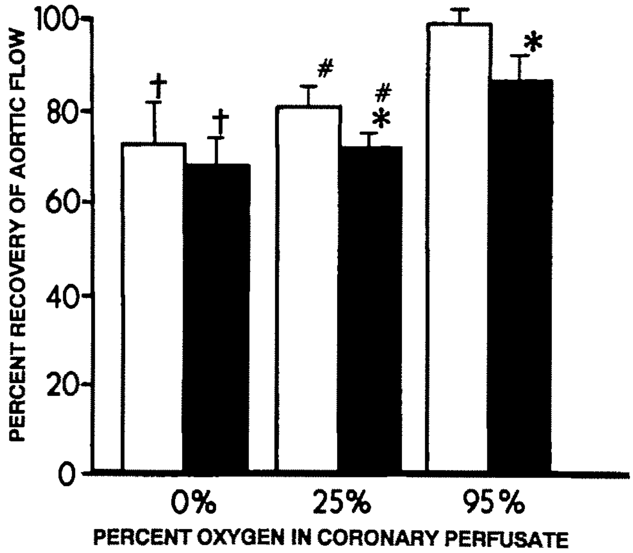

Fig 1. Relationship between postischemic recovery of aortic flow and level of oxygen in the preischemic perfusate. Open bars *represent data from hearts protected by hypothermia alone.* Closed bars *represent data from hearts protected by hypothermia plus cardioplegia. Data shown represent the mean ± the standard deviation from eight hearts per group.* († = $p < 0.05$, *0% oxygen versus 95% oxygen;* # = $p < 0.05$, *25% oxygen versus 95% oxygen;* * = $p < 0.05$, *hypothermia alone versus hypothermia plus cardioplegia.)*

Figure 1 with that of hearts protected with hypothermia plus cardioplegia for each of the oxygen saturations investigated. In hearts perfused with 95% oxygen immediately before ischemia, recovery of aortic flow in the group protected with hypothermia alone was 99% ± 2%; recovery was depressed to 86% ± 5% ($p < 0.05$) in the group protected with both hypothermia and cardioplegia. In hearts perfused with 25% oxygen immediately before ischemia, recovery of aortic flow was 82% ± 4% in hearts protected with hypothermia alone; recovery of aortic flow declined to 72% ± 3% ($p < 0.05$) in the presence of the cardioplegic solution. In hearts perfused anoxically (0% oxygen) immediately before ischemia, recovery of aortic flow was 74% ± 9% in hearts protected with hypothermia alone. This was not different from recovery of aortic flow of 69% ± 6% in the presence of the cardioplegic solution. All hearts protected with cardioplegia reverted to sinus rhythm on reperfusion. There were no significant changes in heart rate on reperfusion for any of the preischemic oxygen saturations investigated. Thus, at equal oxygen concentrations in the coronary perfusate immediately before ischemia, protection by hypothermia plus cardioplegia was inferior to that provided by hypothermia alone.

Postischemic Enzyme Leakage

In hearts protected with hypothermia alone, when the preischemic coronary perfusate was saturated with 95% oxygen, creatine kinase leakage during the 15-minute Langendorff reperfusion period was 16 ± 7 IU/g dry weight. When the perfusate was saturated with 25% oxygen before ischemia, postischemic creatine kinase leakage was 22 ± 7 IU/g dry weight, and when the preischemic perfusate was anoxic (0% oxygen), creatine kinase leakage was 34 ± 8 IU/g dry weight. In hearts protected with hypothermia plus cardioplegia, with the preischemic perfusate saturated with 95% oxygen, postischemic creatine kinase leakage was 23 ± 4 IU/g dry weight. With 25% oxygen saturation, creatine kinase leakage was 32 ± 11 IU/g dry weight, and when the preischemic perfusate was anoxic (0% oxygen), creatine kinase leakage was 41 ± 9 IU/g dry weight. These values were not significant within groups of equal oxygen saturations. There was a significant difference between 0% and 95% oxygen saturation ($p < 0.05$) both for hearts protected with hypothermia alone and for hearts protected with hypothermia plus cardioplegia.

Comment

This study demonstrates that at equal oxygen concentrations of the preischemic perfusate, recovery of postischemic function was better in immature rabbit hearts protected with hypothermia alone compared with hearts protected with hypothermia plus cardioplegia. Furthermore, recovery of postischemic function in the immature rabbit heart was directly related to the oxygen content of the preischemic coronary perfusate. Even in the absence of extracellular oxygen *and* cardioplegic protection, the ischemic immature rabbit heart protected by hypothermia alone recovered well after 2 hours. It is apparent that the lower oxygen content of the cardioplegic solution per se is not responsible for the damaging effect of St. Thomas' II solution on the ischemic immature rabbit heart.

Experimental Model

Rabbits were selected for these studies because as we [3] and others [10] have shown, the ischemic immature heart of this species is damaged by the St. Thomas' II cardioplegic solution. The isolated heart model selected for this study allows for aspects of left heart function and metabolism to be effectively studied in the same preparation over a wide range of experimental conditions with the heart subjected to a physiological work load and temperature. In addition, the model has stable mechanical function with perfusate composition well controlled and easily exchanged within seconds.

The limitations of this type of perfusion system are well known. Aqueous solutions free from red cells, protein, and fatty acids are used to perfuse the coronary arteries, and noncoronary collateral flow is absent. Caution must therefore be applied in transposing results obtained using aqueous perfusates in this experimental model to the clinical situation. St. Thomas' II solution was chosen as an example of a crystalloid cardioplegic solution that has been well characterized and that is in current clinical use in pediatric cardiac surgery [2]. Through preliminary studies, we [3] carefully defined the normal in vivo aortic pressure to which hearts are exposed and then matched the hydrostatic pressure of the isolated hearts to the level appropriate for the immature rabbit.

Oxygen and Preischemic Perfusion

The total volume of coronary effluent collected during the period immediately before ischemia was similar between hearts perfused with Krebs buffer and St. Thomas' II solution. This indicated that there was no difference in the total amount of oxygen delivered to the heart before ischemia for both treatment groups, and that no changes in vascular resistance occurred as a consequence of oxygenation of the preischemic perfusate in any of the treatment groups. Oxygenation of Krebs-Henseleit buffer increased the time taken to achieve diastolic arrest, which was always significantly longer in comparison with the cardioplegia group. In contrast, oxygenation of St. Thomas' II solution had no effect on the time to diastolic arrest. This effect is attributed to the elevated potassium concentration in the cardioplegic solution.

Oxygen and Hypothermic Protection

We have shown that in the ischemic immature rabbit heart protected with hypothermia alone, improved recovery of postischemic function and low postischemic enzyme leakage were directly related to the availability of oxygen in the preischemic coronary perfusate. However, even when the preischemic coronary perfusate was anoxic, there was good recovery of postischemic function but moderate leakage of creatine kinase during reperfusion. This observation indicates that the underlying mechanism responsible for the tolerance to ischemia in immature rabbit hearts is not totally dependent on the availability of extracellular oxygen.

A possible mechanism to account for this tolerance to ischemia may be an increased capacity of the immature heart to produce energy anaerobically to maintain cell survival and function. Maintenance of cellular energy status could be achieved either by an increase in substrate level phosphorylation through glycolysis or by transamination of amino acids. In support of this possibility, immature hearts have a greater reliance on glucose as an energy source than mature hearts [11]. Davtyan and colleagues [12] have demonstrated that preservation of the ischemic neonatal pig heart for transplantation can be improved by the addition of glucose to the storage solutions. Julia and associates [13] have shown that substrate level phosphorylation by amino acid metabolism may be a mechanism whereby the immature canine heart can maintain cellular energy levels during ischemia. Future strategies designed to improve myocardial protection in the immature heart may be able to exploit these metabolic characteristics of the immature heart.

Oxygen and Cardioplegic Protection

We have shown that at equal oxygen saturations of the preischemic coronary perfusate, recovery of postischemic function was superior in immature hearts protected by hypothermia alone than in hearts protected by hypothermia plus cardioplegia. This study indicates that the difference in availability of oxygen in the preischemic perfusate is not solely responsible for the previously documented damaging effect of this cardioplegic solution on the ischemic immature rabbit heart. The identity of the damaging components of the cardioplegic solution remains to be established.

Our results are in contrast with those of Lynch and co-workers [14], who were unable to demonstrate differences in functional recovery between immature rabbit hearts protected with oxygenated Krebs solution and a modified St. Thomas' cardioplegic solution. In their study, hearts were subjected to normothermic ischemia, whereas in our study, the myocardial temperature was maintained at 14°C throughout the ischemic period. This temperature is within the targeted range achieved clinically during cardioplegic protection [2, 15]. The damaging effect of St. Thomas' II solution on the ischemic immature rabbit heart has been confirmed by Kempsford and Hearse [10] to be manifest only during conditions of hypothermia achieved clinically. We therefore question the clinical relevance of studies carried out under normothermic conditions [16] or even less profound hypothermic ischemia [17] to study myocardial protection with a cardioplegic solution in the immature heart. Studies designed to investigate myocardial protection with cardioplegic solutions should be performed under clinically relevant conditions of hypothermia.

Hypothermia influences a number of cellular processes during myocardial ischemia that can delay the onset of cellular injury. One of the major consequences of myocardial cooling is a reduction in cellular oxygen consumption and subsequent metabolic activity of the heart. In studies on myocardial oxygen consumption in the neonatal pig heart, Jessen and associates [18] observed a substantial reduction in oxygen consumption on cooling of the heart, but no further reduction in oxygen consumption on cold hyperkalemic arrest. This observation may partially explain the absence of improved myocardial protection of the immature heart with cold St. Thomas' II cardioplegic solution in comparison with cooling alone.

Postischemic Enzyme Leakage

In this study, low postischemic enzyme leakage was associated with better recovery of postischemic aortic flow (0% oxygen versus 95% oxygen in both treatment groups). This observation is consistent with a previous study of ours [4] that demonstrated that functional recovery is related to postischemic enzyme leakage.

Control of Extracellular pH

As described by the Henderson-Hasselbalch equation, the pH of a bicarbonate-containing solution is partially determined by its carbon dioxide content. We carefully controlled the pH of the preischemic perfusates by gassing the solutions with carbon dioxide. Failure to do so would have decreased dissolved carbon dioxide in the perfusate, thus resulting in a highly alkaline solution. Hendren and co-workers [19] have demonstrated that saturation of a cardioplegic solution with oxygen in the absence of carbon dioxide can blunt the potentially protective effects of oxygenation. We did not correct for the decline in pH of Krebs-Henseleit bicarbonate buffer that occurs with decreasing temperature [20]. By allowing this temperature-induced change to occur, we were able to compare the results of the present study with those of our previous investigations [3, 4] by maintaining identical concentrations of the ionic components of the preischemic perfusates.

Cardioplegia and the Ischemic Immature Heart

Our study demonstrates that the St. Thomas' II cardioplegic solution damages the ischemic immature rabbit heart. This finding is in contrast to the study of Konishi and Apstein [21], who showed that in the isolated, blood-perfused, 4- to 6-day-old rabbit heart, postischemic recovery of ventricular function after protection during ischemia with hypothermia plus St. Thomas' cardioplegia was superior to that provided by hypothermia alone. However, in their study, lidocaine hydrochloride, which is normally excluded from St. Thomas' II solution, was included at a concentration of 0.9 mmol/L, and the sodium content of the solution was reduced from its normal value of 120 mmol/L to 110 mmol/L. These alterations in the composition of the components of the cardioplegic solution, in conjunction with the different experimental model used and the younger age of the group studied, may be responsible for the discrepancy between our two studies. Effective comparisons between studies can be made only if these variables can be standardized between laboratories before investigation.

Conclusion

Although an increased preischemic oxygen content improved recovery of postischemic function in the immature rabbit heart, the lower oxygen content of St. Thomas' II cardioplegic solution compared with the same oxygen content in Krebs buffer was not responsible per se for its damaging effect on the ischemic immature rabbit heart. We conclude that factors other than oxygen availability are responsible for the damaging effect of St. Thomas' II solution on the ischemic immature rabbit heart. Myocardial ischemia results in the adverse redistribution of cellular electrolytes, especially calcium, between the extracellular and intracellular compartments as well as within the organelles of the myocardial cell. Strict control of cellular calcium is an important factor in cardioplegic protection. This is achieved by manipulation of the concentration of the components of the cardioplegic solution. The high calcium concentration (1.2 mmol/L) and alkaline pH (7.8) of St. Thomas' II solution may be inappropriate to prevent intracellular calcium loading of the ischemic immature heart and its subsequent impact on postischemic recovery. We are currently investigating whether the existing calcium and hydrogen ion concentrations of the St. Thomas' II solution are responsible for its damaging effect on the ischemic immature heart.

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Five-Year Results of Coronary Bypass Grafting for Patients Older Than 70 Years: Role of Internal Mammary Artery

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Despite numerous references to the superiority of the internal mammary artery (IMA) over the saphenous vein for myocardial revascularization, its role in the elderly is still in question. From January 1984 through December 1988, 1,081 patients older than 70 years (mean age, 74.9 years) underwent bypass grafting, 354 (33%) receiving left IMA grafts based on the surgeon's preference and 727 (67%) receiving vein grafts only. Selection bias resulted in a higher incidence of known risk factors (such as cardiomegaly, arrhythmias, left ventricular failure, wall motion abnormalities, and preoperative combined New York Heart Association/Canadian Cardiovascular Association functional class IV) in patients in whom the IMA was not used. However, unstable angina, acute myocardial infarction, left ventricular dysfunction, and left main disease were not contraindications for using IMA grafts. The operative mortality rate was significantly lower in IMA patients (2.8% versus 7.6%). The actuarial 5-year survival rate (standard error) was higher in patients with IMA grafts, 89% (3%) versus 78% (2%), and postoperative functional class improved to a greater extent in IMA patients (87% of patients were in classes I and II). Arrhythmias and myocardial infarction were significant causes of late death only in patients with vein grafts. When patients are older than 70 years, patient selection factors clearly play an important role in the differential results between patients in whom the IMA is used and patients in whom vein grafts are used. As in younger patients, excellent results can be achieved in the elderly.

(Ann Thorac Surg 1990;50:940–5)

Direct myocardial revascularization occupies an important role in the management of symptomatic coronary artery disease. Originally, autologous greater saphenous vein was the standard conduit for grafting. In the early 1970s, the internal mammary artery (IMA) was suggested as an alternative in patients whose saphenous vein either was diseased or had been used in a previous operation [1–4]. Numerous studies [1, 2, 4–9] have demonstrated that vein grafts in the aortocoronary position are more vulnerable to atherosclerosis than pedicled internal mammary grafts, and that early and late patency rates are superior for internal mammary grafts.

Nevertheless, many concerns still exist for patients with unstable angina, acute myocardial infarction, arrhythmias, and age greater than 70 years [3, 10–12]. Because the elderly portion of our population is increasing, a larger number of elderly patients with symptomatic coronary artery disease are considered candidates for coronary artery bypass grafting. Past reports [10, 13–15] have documented that this procedure can be performed with acceptable operative mortality and excellent late results in patients older than 70 years.

However, apprehension about the adequacy of flow of the IMA, greater difficulty in pedicle preparation and performance of the anastomosis, and increased risk for a variety of perioperative complications has resulted in the preferential use of saphenous vein grafts in the elderly [3, 11, 13, 16]. This study compares the results of coronary bypass grafting with IMA grafts only or vein grafts only in patients older than 70 years to determine the effect of each conduit on early and intermediate-term survival and relief of symptoms.

Material and Methods

From January 1984 through December 1988, 1,081 patients aged more than 70 years underwent coronary artery bypass grafting at St. Vincent Hospital and Medical Center, Oregon Health Sciences University. This study includes patients with prior coronary bypass procedures but excludes patients who had valve replacement or valve repair. Three hundred fifty-four patients (33%) received left IMA grafts based on surgeon preference, and 727 (67%) received vein grafts only (non-IMA patients). The use of the IMA in coronary artery bypass grafting is no longer restricted to younger patients at our institution. A continually increasing number of elderly patients have received IMA grafts since 1984 (Fig 1). The clinical profile of these patients is shown in Table 1.

The frequency of coronary risk factors was not significantly different between IMA and non-IMA patients

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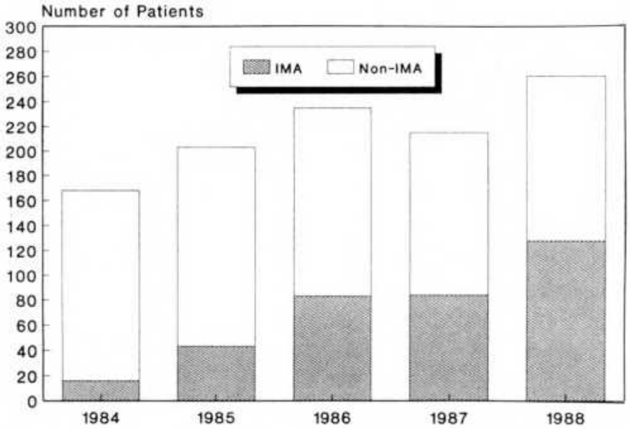

Fig 1. Distribution by year of operation of patients older than 70 years having coronary artery bypass grafting. (IMA = *internal mammary artery;* Non-IMA = *vein grafts only.)*

Table 2. Coronary Artery Disease Risk Factors and Other Preoperative Variables

Variable	IMA Group (%)	Non-IMA Group (%)	*p* Value
Hyperlipidemia	18	13	0.05
Family history of CAD	35	31	0.2
Hypertension	55	51	0.3
Diabetes	17	18	0.9
Cardiomegaly	5	11	0.005
Vascular disease	27	30	0.3
Prior myocardial infarction			0.4
One	31	30	
Two or more	5	7	

CAD = coronary artery disease; IMA = internal mammary artery; Non-IMA = vein grafts only.

(Table 2). Vascular disease included angiographic or Doppler evidence of substantial peripheral vascular or carotid artery disease, or a patient history of cerebrovascular accident.

Left ventricular failure, arrhythmias, or complications during cardiac catheterization were relative contraindications to IMA grafting, whereas the presence of acute myocardial infarction or unstable angina did not prevent use of the mammary artery in elderly patients (Table 3). Chronic/stable angina was defined as medically controlled angina. Unstable angina included rest pain, recent-onset angina, and postinfarction angina. Rest pain was angina at rest that was severe enough to require admission to the hospital, but was not associated with acute myocardial infarction. Recent-onset angina was progressive anginal pain of less than 30 days duration before operation that was increasing in severity, duration, or frequency despite medical treatment. Postinfarction angina was continuing pain after acute myocardial infarction and subsequent operation within 30 days after infarction. The majority of IMA patients were in combined New York Heart Association/Canadian Cardiovascular Association functional class III preoperatively, whereas significantly more ($p <$ 0.01) non-IMA patients were in class IV (Fig 2).

Preoperative catheterization results are shown in Table 4. Angiograms were not available for 161 patients (15%), primarily because they had been returned to the referring cardiologist, and thus, details could not be confirmed at the time of initial data entry into the computer. In a small number of patients, the left ventricular angiograms could not be evaluated, primarily because of an arrhythmia that occurred during the ventriculographic study.

Assessment of left ventricular function was made from angiograms with evaluation of segmental wall motion abnormalities by the Coronary Artery Surgery Study (CASS) method [17]. Five segments in the right anterior oblique view were scored from 1 (normal motion) to 6 (an aneurysm). If all segments were normal, the wall motion score was 5. Mild ventricular impairment was defined by a score of 6 to 10, moderate ventricular impairment included a score of 11 to 15, and scores greater than 15 indicated severely abnormal ventricular function. How-

Table 1. Clinical Data for Coronary Bypass Patients Older Than 70 Years: 1984–1988

Variable	IMA Group	Non-IMA Group
No. of patients	354	727
Male sex (%)	69	67
Age (y)		
Mean	74.1	75.4
Maximum	93	90
Previous coronary bypass grafting (%)	3	4
Follow-up		
Total (patient-years)	469	1,432
Mean (y)	1.3	2.0
Completeness (1988–1989) (%)	93	91

IMA = internal mammary artery; Non-IMA = vein grafts only.

Table 3. Indications for Coronary Artery Bypass Grafting

Indication	IMA Group (%)	Non-IMA Group (%)	*p* Value
Angina			0.003
Chronic/stable	55	45	
Unstable	34	40	
Acute myocardial infarction	7	9	0.4
Arrhythmia	1	2	0.5
Left ventricular failure	1	3	0.07
Catheterization complication	1	3	0.01
Other	2	1	0.1

IMA = internal mammary artery; Non-IMA = vein grafts only.

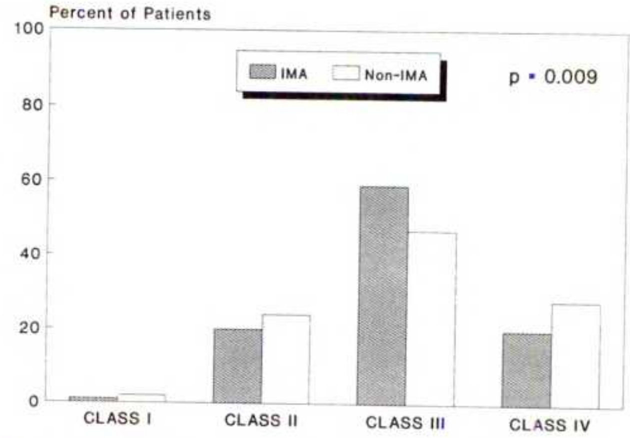

Fig 2. Preoperative functional class. (IMA = *internal mammary artery;* Non-IMA = *vein grafts only.)*

ever, as this scoring system is a subjective assessment for relatively low abnormal scores, the left ventricular end-diastolic pressure was also taken into consideration before left ventricular function was characterized as abnormal. Therefore, left ventricular function was considered abnormal if the left ventricular end-diastolic pressure was 15 mm Hg or higher or if either generalized or segmental wall motion abnormalities were present.

Major coronary artery stenosis was defined as 50% or greater narrowing of the diameter of the vessel. Left main coronary artery disease did not preclude use of the IMA, as it was present in 12% of IMA patients versus 17% of non-IMA patients.

Operative Technique

After a median sternotomy, the IMA and accompanying veins were dissected as a pedicle, going proximally beyond the first intercostal branch and distally after the bifurcation into the superior epigastric and musculophrenic arteries. In all instances, branches were controlled with clips in the mammary side and with diathermy in the chest wall. The pleura was routinely entered to provide a more direct route to the coronary artery and to avoid medial displacement by the lung. The vessels were stored undivided substernally in a papaverine hydrochloride–soaked sponge.

Table 4. Preoperative Catheterization Data[a]

Variable	IMA Group	Non-IMA Group	*p* Value
Wall motion abnormality	36	46	0.007
Mean LVEDP (mm Hg)	11.8	13.7	0.008
LV function			0.1
Normal	44	37	
Abnormal	56	63	
Diseased vessels			0.6
One	7	9	
Two	17	18	
Three	40	36	
Four or more	36	37	
Left main disease	12	17	0.04

[a] Data are shown as percentages unless otherwise indicated.

IMA = internal mammary artery; LV = left ventricular; LVEDP = left ventricular end-diastolic pressure; Non-IMA = vein grafts only.

After the dissection of the mammary artery, cardiopulmonary bypass was instituted, and the body temperature was lowered to 30°C. The vein grafts were inserted first. The distal anastomoses were performed during a brief period when the aorta was cross-clamped and the heart fibrillated. The proximal vein anastomoses were made with the heart beating. Cardioplegia was not commonly used for myocardial protection. The IMA was inspected before its anastomosis for evidence of injury and adequacy of blood flow.

Emergency operation warranted by an unstable hemodynamic condition was performed in 14% of the non-IMA patients compared with 1% of the IMA patients. The operation was carried out on an elective or urgent basis in 77% and 8% of non-IMA patients compared with 92% and 6% of IMA patients, respectively.

Data Management

Detailed data were collected on all patients. Prospective annual follow-up by questionnaire and telephone was conducted by experienced research assistants. Patients were followed up on a continual basis, independent of any particular review for publication. All patient complications were confirmed by medical records, and causes of death were determined from autopsy reports, final summaries, or death certificates. All follow-up data were computerized for subsequent analysis.

Survival probabilities and standard errors were estimated by the actuarial method [18]. Actuarial curves were compared by means of the Lee-Desu statistic [19]. Chi-square statistics were used to compare categorical variables, and analysis of variance was used for continuous variables. A *p* value of less than 0.05 was considered significant.

Results

Operative mortality included all deaths within 30 days of operation and those that occurred more than 30 days after operation if they were a direct outcome of the procedure. The operative mortality rate was significantly lower ($p = 0.003$) in the patients with IMA grafts, 2.8% versus 7.6% for non-IMA patients. The causes of operative death are shown in Table 5. Noncardiac causes of death included pulmonary embolus, respiratory failure, pneumonia, multiple-system failure, renal failure, cerebrovascular accident, bowel infarction, and sepsis.

The occurrence of perioperative myocardial infarction was similar in the two groups, 1.4% for IMA patients and 1.9% for non-IMA patients. No significant difference ($p = 0.08$) in mean hospital stay was found between the two groups: 10.7 days for IMA patients versus 11.8 days for non-IMA patients.

Table 5. Causes of Operative Death

Cause	IMA Group	Non-IMA Group
Cardiac related		
LV failure	2	12
Arrhythmia	1	10
Myocardial infarction	1	5
Dissection of aortotomy site	0	1
Ruptured aorta	1	2
Tamponade	0	1
Noncardiac	4	23
Cause pending	1	1
Total	10 (2.8%)	55 (7.6%)

IMA = internal mammary artery; LV = left ventricular; Non-IMA = vein grafts only.

Table 6. Causes of Late Death

Cause	IMA Group	Non-IMA Group
Cardiac related		
Congestive heart failure	1	8
Arrhythmia	2	9
Myocardial infarction	0	5
Sudden death	2	6
Noncardiac	4	19
Cause pending	4	8
Total	13 (2.8%/ patient-year)	55 (3.8%/ patient-year)

IMA = internal mammary artery; Non-IMA = vein grafts only.

The actuarial 5-year survival (standard error) was significantly higher ($p = 0.001$) for IMA patients than for non-IMA patients, 89% (3%) versus 78% (2%) (Fig 3). Arrhythmia and myocardial infarction were significant causes of late death in non-IMA patients only (Table 6). Noncardiac causes of late death included chronic obstructive pulmonary disease, respiratory failure, cerebrovascular accident, bowel infarction or obstruction, sepsis, hepatitis, cancer, and accidental causes.

Actuarial freedom from reoperation at 5 years was 99% (1%) for both groups. Incidence of postoperative myocardial infarction was not significantly different ($p = 0.7$) between the two groups: 98% (1%) for IMA patients and 97% (1%) for non-IMA patients.

Significant postoperative improvement in symptomatology was found in both groups (Fig 4). The majority of patients returned to combined New York Heart Association/Canadian Cardiovascular Association functional class I or II. In the IMA group, 73% of patients were in class I and 14% in class II, whereas for non-IMA patients, the corresponding percentages were 67% and 17%.

Comment

As the life span of humans continues to increase and as the standard of health care generally improves, more people are reaching advanced age and are seen with problems warranting particular attention. Symptomatic coronary artery disease represents a great percentage of the heart disease in elderly patients. In the past decade, many reports [3, 14, 15, 20] have documented that coronary artery grafting can be performed in the elderly with acceptable operative mortality and favorable long-term results. Soon after coronary bypass surgery was established as a clinical reality, it was demonstrated that vein grafts in the aortocoronary position were far more vulnerable to atherosclerosis than pedicled IMA grafts [1, 2, 9, 21]. Reports of excellent 7- to 10-year patency rates for

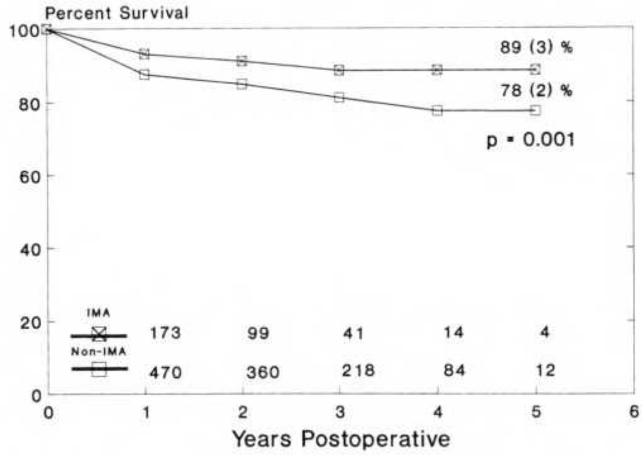

Fig 3. Survival after coronary artery bypass grafting. (IMA = *internal mammary artery;* Non-IMA = *vein grafts only.)*

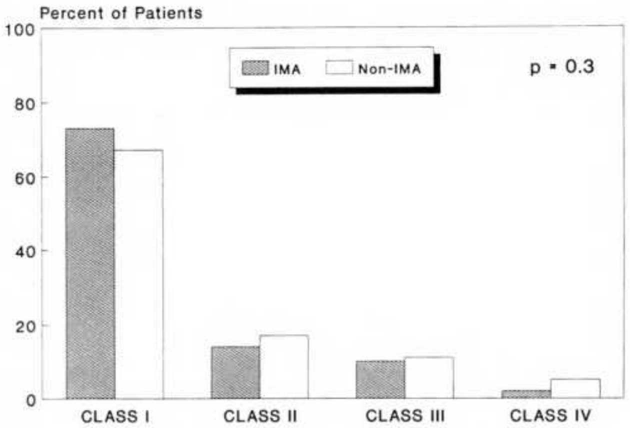

Fig 4. Postoperative functional class. (IMA = *internal mammary artery;* Non-IMA = *vein grafts only.)*

IMA grafts and the absence of atherosclerosis have been published [2, 4, 5, 7, 11, 21, 22]. Ten-year patency of mammary grafts ranges from 69% to 95% in the reports of Barner [2], Tector [5], Okies [21], and their colleagues.

Apart from the increased long-term patency rates, the IMA carries a decreased need for reoperation and fewer late cardiac events. Loop and associates [7] found that patients who received only vein grafts had a 1.61 times greater risk of death in a 10-year period compared with those who had grafting with the IMA. In addition, patients with only vein grafts had 1.41 times the risk of late myocardial infarction, 1.25 times the risk of hospitalization for cardiac events, 2 times the risk of cardiac reoperation, and 1.27 times the risk of all late cardiac events compared with those with IMA grafts.

However, despite the numerous references to the superiority of the IMA over the saphenous vein for myocardial revascularization, its role in the elderly is still in question. Many cardiovascular surgeons are concerned about the short projected longevity of elderly patients, left main coronary artery stenosis, left ventricular dysfunction or hypertrophy, acute myocardial infarction, arrhythmias, and unstable hemodynamics [3, 11, 13, 16, 23].

Severe hyperlipidemia and diabetes mellitus are regarded as relative contraindications for IMA grafting because of the risk of developing earlier atherosclerotic changes in the brachiocephalic artery and IMA after the operation. Similar apprehension exists concerning excessive stretching of the grafts by emphysematous lungs [24–26]. Reports by Tsai [13], Rich [10], and their coworkers show increased perioperative complications and hospital mortality in patients with heart failure, acute ischemic syndromes, and left ventricular dysfunction and patients in functional class IV.

In this series, selection bias resulted in a higher incidence of known risk factors in the non-IMA group, risk factors such as cardiomegaly, arrhythmias, left ventricular failure, wall motion abnormalities, preoperative functional class IV, and emergency operation. However, unstable angina, acute myocardial infarction, left ventricular dysfunction, and left main disease were not contraindications for IMA grafting.

The operative mortality rate has ranged from 0% to 14% in patients older than 70 years undergoing coronary artery bypass grafting alone, with the larger series and those including patients less than 80 years old generally reporting lower mortality rates [2, 3, 10, 13–15, 21]. The low hospital mortality rate of 2.8% for patients receiving IMA grafts in this series is comparable with that in younger patients undergoing coronary bypass procedures. This supports the idea that bypass grafting should be offered to older patients when the proper clinical indications are present.

Knowing there is no direct measure of adequacy of myocardial perfusion by bypass grafts, surgeons must rely on indirect clinical variables, such as operative and late deaths and early and late complications [9]. Although the preoperative anatomical and pathophysiological status of the individual patient bears on late results, indirect indications of IMA graft patency in this series were the significantly lower operative and late mortality rates and the fewer fatal arrhythmias and myocardial infarctions in the early and late postoperative periods. This relative immunity against arrhythmias and myocardial infarctions that the IMA graft gives to patients, together with the better intermediate-term palliation (87% of patients with IMA grafting were in class I or II postoperatively) advocates a high patency rate.

In the CASS study [17], the reported duration of hospital stay for a bypass grafting procedure was significantly longer ($p < 0.001$) for patients 65 years old or older (13.3 ± 0.2 days) compared with that for patients less than 65 years of age (11.4 ± 0.4 days). The duration of hospital stay for patients in our series was comparable with that for much younger patients reported in other studies [10, 20, 27].

There is little that can be done to upgrade the excellent 5-year survival rate of 89% in patients with IMA grafts, which is similar to the expected survival of the "normal" population in the same age group and which was significantly higher ($p = 0.001$) than the survival rate for non-IMA patients (78%). Higher 5- and 10-year longevity after IMA grafting has been reported, but in patients of younger age by at least one decade [2, 4, 11, 21, 27].

Routine use of the mammary artery did not show any increase in the incidence of major surgical complications in patients older than 70 years, even in the presence of diabetes, according to Gardner and colleagues [28]. The same investigators showed a significantly better survival of 87% at 4 years in patients receiving IMA grafts compared with 77% for those with vein grafts only.

However, therapeutic considerations for patients of advanced age should focus less on survival as an objective and more on quality of life and symptomatic relief. Even in these categories, use of the IMA carries superior results, as shown by the better postoperative functional class of the patients in this series.

An important question that arises relates to the selection process in elderly patients considered candidates for IMA grafting. Controversy still exists among surgeons on the role of various known risk factors. Therefore, patient selection must be based on available data and clinical judgment.

In this series, we have shown that patient selection factors clearly play an important role in the differential results between IMA and non-IMA patients older than 70 years. As in younger patients, excellent results can be achieved in elderly patients undergoing coronary artery bypass grafting.

We appreciate the assistance of Andrew J. Bennett in the preparation of the manuscript.

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Morphological and Histopathological Aspects of Aneurysms After Patch Aortoplasty for Coarctation

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Repair of coarctation of the aorta by synthetic patch grafting has been complicated by late aneurysm formation. These aneurysms differ macroscopically from atherosclerotic thoracic aortic dilatations. Specimens for microscopic examination were taken from 14 of 20 patients undergoing aneurysm resection. Histological analysis of the specimens showed medionecrosis in 13 patients (93%), foreign body reaction in 11 patients (78%), and intimal thickening in 3 patients (21%). The three layers of the aortic wall could be identified in the aneurysms. On the basis of these results, we discuss the etiologic factors and pathogenetic mechanisms involved in the development of these aneurysms.

(Ann Thorac Surg 1990;50:946–8)

Correction of aortic coarctation with the technique of prosthetic patch aortoplasty involves a high rate of late aneurysms that develop at the site of repair. Typically, the aneurysms form adjacent or opposite to the synthetic patch years after the primary operation. We and other authors have reported the results of operative treatment of this unexpected complication [1–8]. However, with the exception of a few microscopic examinations of occasional patients, we have not found studies of the structure of this special type of aneurysm. In this report, we give a more detailed morphological description of the aneurysms operated on and the results of microscopy. The possible pathogenetic mechanisms in the development of these late aneurysms are discussed in the light of the present results.

Material and Methods

Synthetic patch (Dacron or Teflon) aortoplasty was used as an alternative method in the repair of coarctation of the aorta in 67 adult patients between 1967 and 1978. The technique used has been described by Vossschulte [9]. Anastomoses were made with nonabsorbable synthetic suture material. The mean age of the patients at primary repair was 25.3 years. Twenty-three patients (34.3%) have undergone reoperation because of subsequent aneurysm development at the repair site. Seventeen patients were male and 6 were female. Their mean age at reoperation was 37.7 years. As to the long-term results of reoperations, we refer to previous reports from our hospital [1, 2]. In 20 patients, the aneurysmal sac was incised longitudinally, and the aorta was reconstructed with a tubular vascular prosthesis using the inlay technique: the aneurysmal sac was sutured around the prosthesis to isolate it from the surrounding tissues. Three patients were treated by external support of the aneurysm with Dacron fabric.

Specimens from the incised border of the aneurysm could be taken in 14 patients. The resected specimens were fixed in formalin and embedded in paraffin. For microscopic study, the specimens of the resected aneurysm were stained by hematoxylin and eosin, Weigert elastin, van Gieson, and periodic acid-Schiff and were analyzed using an Olympus light microscope.

Results

The size of the aneurysms varied from a small bulge to a dilatation 10 cm in diameter. Figures 1 and 2 show a typical example. Adjacent pleural and lung tissues were always found to adhere to the aneurysm as a consequence of scarring after the primary repair. The aneurysms were formed either from a single pouch or most frequently from several bulges appearing as bluish, translucent swellings. The walls were brittle and thin, and there was no advanced atherosclerosis. The patches and suture lines were intact.

In microscopic studies, all 14 resected aortic specimens showed histopathological changes. All layers of the wall could be identified. The most common histological finding was necrosis of the smooth muscle cells associated with loss and fragmentation of elastic fibers. These medionecrotic changes were found in 13 patients (93%). A foreign body reaction was seen in 11 specimens (78%) often near the patch and the suture materials. Atherosclerotic intimal thickening was found in 3 patients (21%). Small patches of smooth muscle cells were often seen within the fibrotic wall confirming that these formations were true aneurysms (Fig 3). Nonspecific aortitis was not found.

Comment

Anastomotic aneurysms or the aneurysms between synthetic fabric and arterial wall are usually considered false

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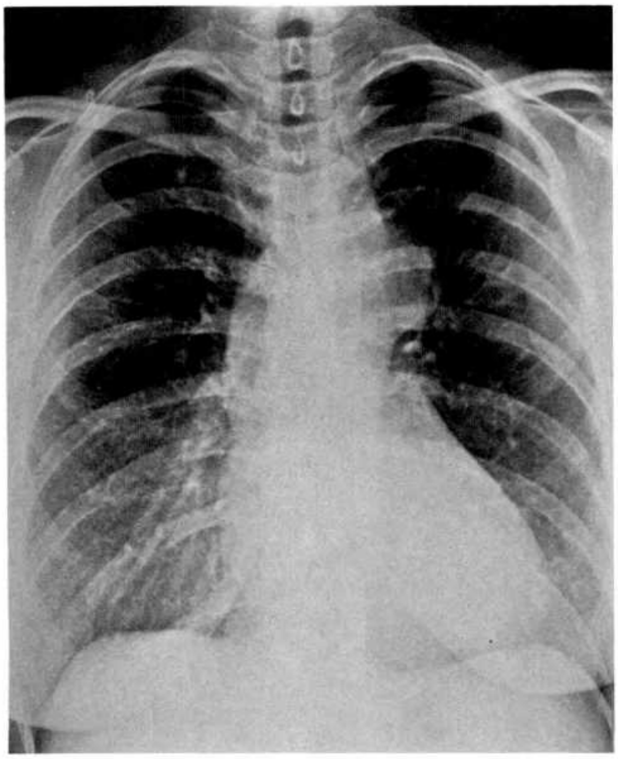

Fig 1. Chest roentgenogram showing a large aneurysm 17 years after patch grafting for aortic coarctation. The aortic arch is prominent.

aneurysms. The bulges described after patch grafting for coarctation repair have been both false and true aneurysms [3, 4, 6, 8]. In our patients, all aneurysms were true aneurysms as opposed to anastomotic false aneurysms. Microscopic survey showed that elements of aortic media could be identified in the aneurysm wall. Our results suggest that aneurysms developing after patch grafting of aortic coarctation may commonly, if not always, be true aneurysms. In addition, the integrity of the suture lines indicates that these particular bulges constitute a normal type of aneurysm.

Most aneurysms of the descending thoracic aorta result from advanced atherosclerosis. In our study, the aneurysms were not typical atherosclerotic dilatations because there was no macroscopic calcification or other signs of advanced atherosclerosis. Intimal thickening found in three specimens (21%), however, gave evidence of premature atherosclerosis in these relatively young patients.

We agree with other authors that there is no infectious background to these aneurysms [3, 6]. With the exception of one mycotic pseudoaneurysm [10], a contribution by inflammatory disorders in aneurysm development after corrective operation for isthmic aortic coarctation with synthetic patch aortoplasty has not been reported.

Histopathological changes found in aneurysms and in the aortic wall of the patients undergoing synthetic onlay patch aortoplasty for aortic coarctation have been due to degeneration of the media, loss and fragmentation of elastic material, and advanced fibrosis [3, 5–7]. Medionecrosis, which is obviously a factor contributing to aneurysm formation by weakening of the vessel wall, was present in more than 90% of our patients. Unlike in the microscopic analysis of Bergdahl and Ljungqvist [3], evidence of foreign body reaction was present in 11 of the 14 specimens in our study.

Pathogenesis of the aortic dilatation after patch aortoplasty for coarctation has remained obscure. Unlike anastomotic false aneurysms, these bulges seem to arise beside or opposite the synthetic patch, finally involving the whole aortic circumference. Rigid prosthetic material implanted into the aorta creates local hemodynamic tension and abnormal turbulence against the vessel wall. Differences in compliance between the aortic wall and the inelastic patch have previously been suggested by us and other authors as the most important factor in aneurysm formation [1–4, 11].

In conclusion, the results of the microscopic analysis in the present study seem to further elucidate the former hypothesis, thus confirming our earlier reports. Patients with aortic coarctation seem to have an inherent weakness of the aorta or the segment of the aortic wall owing to medial cystic degeneration. The medionecrosis, in addition to the foreign body reaction after operation, might form a pathogenetic basis for the local dilatation and the aneurysm development. An aortic wall weakened by congenital or acquired medial degeneration is less resistant, and it dilates in response to hemodynamic forces resulting from lack of compliance between the vessel wall and the prosthesis and from foreign body reaction caused by the prosthetic and suture materials.

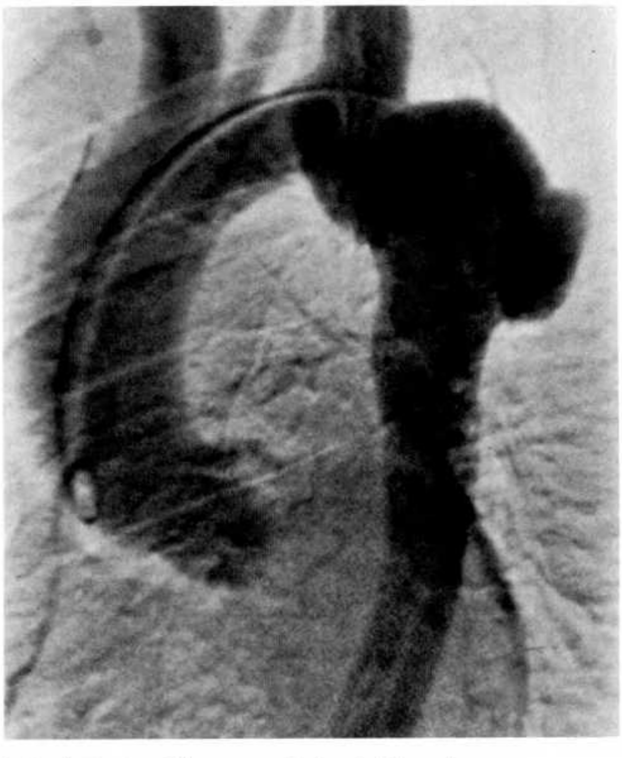

Fig 2. Aortogram of the same patient as in Figure 1.

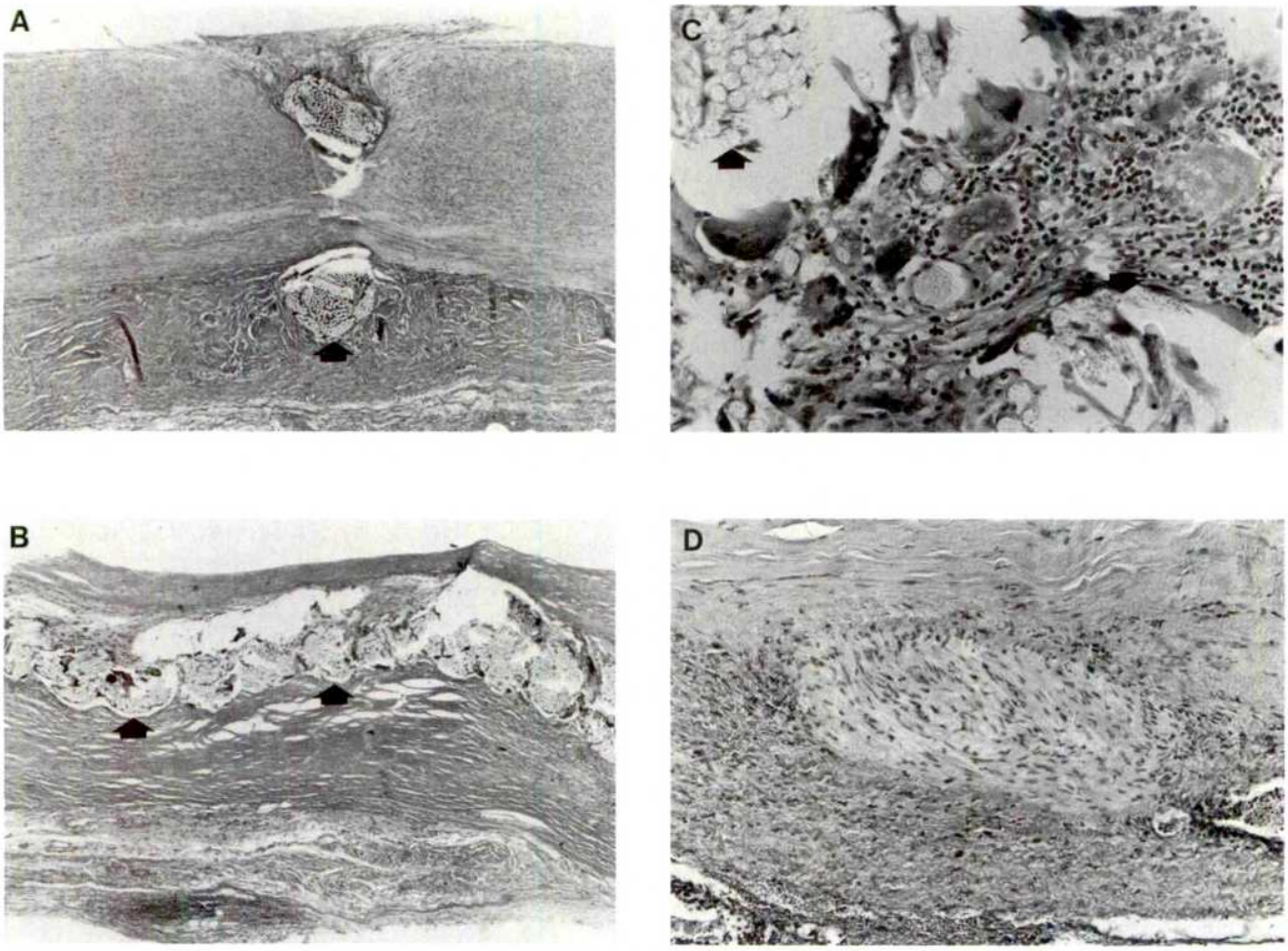

Fig 3. Histopathological findings of the aneurysms. The arrows *show the suture and patch materials. (A) Suture causing local scarring in an otherwise well-preserved wall of the aneurysm. (B) Low magnification of the aneurysm shows abundant scarring of the aortic wall (medionecrosis), intimal thickening with some foam cells, and a foreign body reaction around the patch material. (C) Foreign body reaction was a constant finding in all samples containing either suture or patch material. (D) A cluster of normal medial smooth muscle cells trapped within the fibrous tissue of the aneurysm. (Hematoxylin and eosin, ×30 before 19% reduction [A, B], ×350 before 19% reduction [C], and ×200 before 19% reduction [D].)*

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Sternotomy Infection: Poor Prediction by Acute Phase Response and Delayed Hypersensitivity

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Two hundred twenty-one consecutive adult cardiac surgical patients were examined prospectively for nutritional protein state, acute phase protein response, and delayed hypersensitivity reaction in an attempt to identify patients at high risk for the development of sternal wound infection, which occurred in 6 patients (2.7%). There was no significant correlation between preoperative nutritional protein concentrations (retinol-binding protein, prealbumin, and transferrin) and acute phase protein levels (C-reactive protein, α_1-acid glycoprotein, and complements B and C_3), nor a statistically significant relationship between nutritional state or acute phase protein response and the development of sternal infection. Preoperative complement C_3 levels were elevated, however, in 80.0% of those in whom sternal infections developed compared with 30.6% of those with well-healed wounds. Similarly, postoperative concentrations of α_1-acid glycoprotein were elevated in 80.0% of those in whom sternal infections developed compared with 28.6% of those with well-healed wounds. There was no correlation between delayed hypersensitivity and the risk of sternal infection, nor between preoperative nutritional protein and acute phase protein values. Seventy-three percent of patients were anergic on postoperative day 2. Stepwise logistic regression showed that age, body weight, preoperative intensive care unit stay, repeat median sternotomy, internal mammary artery grafting, postoperative hemorrhage, and postoperative cardiac arrest correlated with the development of sternal infection, whereas transfusion requirement, reexploration for bleeding, and the operation performed did not. We conclude that routine delayed hypersensitivity testing is of no value in predicting high-risk cardiac surgical patients when the anergy battery is placed on the preoperative day. Although statistically insignificant, possibly due to the small number of patients in whom sternal infection developed in this study (type II error), a larger study might find preoperative complement C_3 and postoperative α_1-acid glycoprotein levels to be predictive of patients at risk for the development of sternal wound infection. The final logistic model for the predicted risk (%) of sternal wound infection is: PREDSWC = exp(EQ)/1 + exp(EQ) where EQ = (0.38 × age) + (0.24 × weight) + (5.42 × preop ICU) + (4.39 × redo) + (7.14 × IMA) + (4.49 × hemorrhage) + (8.81 × arrest) − 62.72, and where preop ICU, redo, hemorrhage, and arrest are defined as yes (1) or no (0), IMA is defined as 0, 1, or 2, age is in years, and weight is in kilograms.

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Sternal wound infection after cardiac surgical procedures results in nearly a doubling of postoperative hospitalization, a substantially increased cost, and limited capacity for productive activity if the patient survives [1]. Previous studies, mostly retrospective in nature, have not clearly delineated specific risk factors for the development of sternal infection, so that modification of such risk factors might be undertaken in the preoperative period.

Most risk factors for the development of wound infection in the general surgical population have been well defined [2] and often relate to the patient's concurrent medical problems, nutritional status, and immune state. Such risk factors for cardiac surgical patients are less well defined [1, 3–5]. Obesity, diabetes mellitus, and chronic obstructive pulmonary disease are commonly associated with coronary artery disease. Patients with severe chronic illnesses such as renal failure, collagen vascular disease, and myeloproliferative diseases are now undergoing cardiac operations [2].

Low levels of the central nutritional proteins such as transferrin, prealbumin-retinol binding protein complex, and albumin are seen with nutritional depletion [6]. The immunologic defects seen in protein and protein-calorie deficiencies in both humans and experimental animals include low levels of individual complement components, impaired opsonic and neutrophil function, impaired delayed hypersensitivity, decreased lymphoid mass and function, and increased susceptibility to infection. A previous study has suggested that sternal wound infection might be predicted early in the postoperative period by the measurement of α_1-acid glycoprotein and C-reactive protein [7].

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Failure of cell-mediated immunity, as determined by anergy in the delayed hypersensitivity response, can be correlated to nutritional state [8, 9] and has been statistically associated with greater morbidity [9, 10], sepsis, and mortality [9] in surgical and posttrauma patients.

The purpose of this study was to prospectively examine adult patients undergoing elective cardiac surgical procedures for clinical or biochemical variables (specifically: nutritional status, acute phase protein response, and delayed hypersensitivity response) that might predict the development of sternal wound infection.

Material and Methods

Two hundred twenty-one consecutive adult cardiac surgical patients admitted to The Christ Hospital between May 1987 and January 1988 were prospectively studied. The protocol was approved by the Institutional Review Board of The Christ Hospital on May 7, 1987, and informed consent was obtained.

Retinol binding protein, prealbumin, and transferrin levels were measured 1 day preoperatively to assess nutritional status. All patients similarly had levels of complement B (properdin) and C_3, C-reactive protein, and α_1-acid glycoprotein measured on the preoperative evening and on postoperative day 2. Quantification was determined using the Radioimmunodiffusion Plate (RID; Kallestad Diagnostics Inc, Austin, TX).

Delayed hypersensitivity responses were determined using intradermal injection of 0.1 mL of each of three recall antigens given on the preoperative evening: mumps, 40 complement fixation units/mL saline solution (Connaught Laboratories Inc, Swiftwater, PA); *Histoplasma capsulatum*, 0.01 parts/mL (Parke-Davis, Division of Warner-Lambert Co, Morris Plains, NJ); and *Candida albicans* protein, 0.002 units/mL (Berkley Biologicals Inc, Berkley, CA). Injection sites were graded at 48 and 72 hours after administration (24 and 48 hours postoperatively) by one of three observers, and were recorded as positive if the resulting induration was greater than or equal to 5 mm in diameter at either reading. Patients were categorized as normally reactive if two of three sites were positive, relatively anergic if one site was positive, and anergic if there was no positive response to any antigen [8, 9].

Cardiac operations were performed using median sternotomy under standardized conditions of membrane oxygenation with moderate (28°C) systemic hypothermia, and myocardial protection using cold potassium crystalloid cardioplegia as well as topical cooling. All patients received 1 g of cephapirin sodium on induction of anesthesia as well as every 6 hours for 48 hours.

Sternal wounds were evaluated in the hospital on a daily basis by the primary surgeon, with follow-up of 16 to 24 months. Any wound requiring drainage or debridement was classified as infected. Wounds were graded as: grade 1, well healed; grade 2, infection of the subcutaneous tissues; grade 3, infection involving the deep tissues exclusive of bone; and grade 4, infection involving bone.

Thirty-eight other variables including age, sex, weight, diabetes mellitus, left ventricular ejection fraction, pack-years smoked, room air oxygen tension, use of preoperative steroids, preoperative hospital and intensive care unit days, preoperative infections and sites, American Society of Anesthesiology (ASA) class, surgeon, operation received, primary or repeat sternotomy, number of internal mammary arteries used, cardiopulmonary bypass and aortic cross-clamp times, wound closure technique, inotropic support and afterload reduction, aortic balloon counterpulsation, transfusion requirement, postoperative hemorrhage with or without reexploration, postoperative cardiac arrest, perioperative myocardial infarction, number of days that chest tubes remained, postpericardiotomy syndrome/treatment, days until restitution of postoperative nutrition, postoperative infections and sites, and postoperative intensive care unit and hospital days were analyzed.

Data were analyzed using a Compaq 386 computer and BMDP statistical software (Los Angeles, CA). The Pearson χ^2 and Pearson product-moment correlations were used to determine statistical significance, defined as p less than 0.05. Variables were then entered into a stepwise logistic regression analysis with a cut-off point set at 50% (BMDP, LR, MLR selection method) to create a model for prediction of the individual patient's risk of sternal infection.

Results

The 221 patients consisted of 162 men (73.3%) and 59 women (26.2%) with a mean age of 60.9 ± 0.7 years (range, 33 to 82 years) and a mean weight of 80.2 ± 0.1 kg (range, 45.4 to 145.0 kg). Forty-four had diabetes mellitus (19.9%) and 15 were insulin dependent (6.8%). There were 119 patients (53.0%) who smoked an average of 51.5 ± 3.0 pack-years (range, 10 to 200 pack-years). Room air oxygen tension was determined in only 28 patients (12.7%) and averaged 77.6 ± 2.3 mm Hg (range, 54.0 to 96.0 mm Hg). Seven patients were taking steroids preoperatively. Left ventricular ejection fraction was estimated using left ventriculography, two-dimensional echocardiography, or multiple gated acquisition ventriculoscintigraphy. The mean left ventricular ejection fraction for 109 patients (49.3%) was 0.55 ± 0.01 (range, 0.20 to 0.70), with 41 (37.6%) having a left ventricular ejection fraction less than or equal to 0.55 and 12 (11.0%) having a left ventricular ejection fraction of 0.30 or less. Clinical severity was judged independently using the ASA classification system: 1.4% (n = 3) were in ASA class II, 63.8% (n = 141) were in ASA class III, and 34.8% (n = 77) were in ASA class IV. One hundred forty-four patients (65.2%) were admitted on the preoperative evening. The remaining 77 patients (34.8%) were hospitalized 2 to 35 days before operation. Thirty-nine patients (17.7%) spent an average of 7.0 ± 0.9 preoperative days in the intensive care unit (range, 1 to 24 days). A preoperative intensive care unit stay significantly correlated with a 29.0% risk of development of a preoperative infection ($p < 0.00001$). Eleven patients had culture-proven preoperative infections (Table 1).

The 221 patients underwent a total of 252 median

Table 1. Preoperative Infections

Site	Organism	N
None	. . .	210
Urine	*Escherichia coli*	4
	Pseudomonas aeruginosa	1
	Enterobacter cloacae	1
	Enterococcus	1
	Lactobacillus	1
Sputum	*Legionella pneumophila*	1
Skin	*Hemophilus influenzae*	1
Hand	*Staphylococcus aureus*	1

sternotomies by six surgeons. The vast majority were for atherosclerotic coronary artery occlusive disease followed by, or combined with, operations for acquired valvular heart disease (Table 2). One hundred eighty-two patients (82.4%) had primary operations whereas 39 had undergone prior median sternotomy, 1 patient twice. A single internal mammary artery was used in 153 patients (69.2%), whereas both internal mammary arteries were used in 9 patients (4.1%). Data on cardiopulmonary bypass time, aortic cross-clamp time, and other interventional support are recorded in Table 3.

Inotropic support and afterload reduction were calculated on the number of drugs multiplied by the number of days on each drug (drug-days). Afterload reduction, consisting of nitroprusside or nitroglycerin, or both, was used in 182 patients (82.4%). One hundred thirty-one patients (59.3%) required inotropic support consisting of various α- and β-adrenergic and dopaminergic agonists. Only 1 patient required use of intraaortic counterpulsation. Four patients received external closed-chest compression for postoperative cardiac arrest and were successfully resuscitated. Postoperative hemorrhage, defined as chest tube output exceeding 150 mL/h for 2 or more hours, occurred in 33 patients (14.9%). Thirty patients (13.6%) required reexploration for postoperative hemorrhage, 1 patient twice. One hundred sixty-six patients required transfusion of an average of 3.2 units of packed red blood cells.

Table 2. Operation Performed

Operation	N	%
CABG	175	79.2
IMA		
Single	153	69.2
Bilateral	9	4.1
AVR/CABG	8	3.6
MVR/CABG	3	1.4
AVR	17	7.7
MVR	8	3.6
AVR/MVR	6	2.7
LVA/CABG	2	0.9
Atrial myxoma	1	0.5
ASD	1	0.5

ASD = atrial septal defect; AVR = aortic valve replacement; CABG = coronary artery bypass grafting; LVA = left ventricular aneurysm; MVR = mitral valve replacement.

Table 3. Perioperative Statistics

Variable	N (%)	Mean ± SEM	Range
Primary operation	182 (82.4)		
Repeat operation	39 (17.7)		
CPB	221 (100.0)	107.9 ± 2.9^a	0 to 284^a
X-clamp	128 (57.9)	66.3 ± 2.3^a	0 to 140^a
Afterload reduction	182 (82.4)	1.4 ± 0.1^b	0.33 to 7.0^b
Inotrope support	131 (59.3)	1.4 ± 0.2^b	0.33 to 14.0^b
IABP	1 (0.5)		
CPR	4 (1.8)		
Hemorrhage	33 (14.9)		
Reexploration	30 (13.6)		
Transfusion (pRBC)	166 (75.1)	3.2 ± 0.2^c	1 to 18^c

[a] In minutes. [b] In drug-days. [c] In units.

CPB = cardiopulmonary bypass; CPR = cardiopulmonary resuscitation; IABP = intra-aortic balloon pump; IMA = internal mammary artery; pRBC = packed red blood cells; SEM = standard error of the mean; X-clamp = aortic cross-clamp.

The majority of patients (n = 163) had their mediastinal tubes removed on postoperative day 2 (range, 2 to 21 days). Parenteral nutrition was instituted if gastrointestinal dysfunction exceeded 5 to 7 postoperative days. The mean length of nutritional deprivation was 1.7 days (range, 1 to 8 days). Pericarditis, diagnosed by an increased erythrocyte sedimentation rate and recurrent or persistent pericardial friction rub, occurred in 41 patients (mean postoperative day = 7.0). All patients with pericarditis were treated with prednisone.

There were five deaths (2.3%) occurring on postoperative days 0, 4, 5, 7, and 16. Four of the deaths occurred in patients classified preoperatively as in ASA class IV, indicating the severity of their illness. Excluding deaths, the mean postoperative intensive care unit stay was 4.2 ± 0.5 days (median, 2.0 days; range, 2 to 83 days) and the average length until discharge was 13.8 ± 0.7 days (median, 11.0 days; range, 6 to 104 days).

Postoperative infections (Table 4) occurred in 53 patients (24.0%) and strongly correlated with the length of postoperative intensive care unit stay ($p = 0.00001$). There was a strong relationship between the postoperative hospital days and both the development of postoperative infection ($p < 0.00001$) and the organism involved ($p = 0.00017$). There was also a positive correlation between preoperative intensive care unit stay and postoperative infection ($p = 0.00001$). However, there was no correlation between the occurrence or site of preoperative and postoperative infection. Furthermore, preoperative infective organism did not affect the organism involved postoperatively.

Sternal wound infections developed in 6 patients for a rate of 2.7%. There were three grade 2 infections; two were culture negative and one grew *Enterobacter aerogenes*.

Table 4. Postoperative Infections and Sites

Organism	Sternum	Urine	Sputum	Leg	Mouth	Valve	Catheter Site	Blood	Total
None[a]	2	. . .	. . .	1	. . .	1	. . .	. . .	172
P aeruginosa	. . .	. . .	1	. . .	. . .	. . .	. . .	1	2
S epidermidis	1	. . .	. . .	. . .	. . .	. . .	4	. . .	5
E cloacae	. . .	1	. . .	. . .	. . .	. . .	. . .	. . .	1
E aerogenes	1	1	1	. . .	. . .	. . .	. . .	. . .	3
S aureus	1	. . .	. . .	. . .	. . .	. . .	1	. . .	2
K pneumoniae	. . .	. . .	1	. . .	. . .	. . .	. . .	. . .	1
E coli	. . .	1	. . .	. . .	. . .	. . .	. . .	. . .	1
Mixed gram-negative	. . .	3	6	. . .	. . .	. . .	. . .	. . .	9
Mixed gram-positive and gram-negative	1	. . .	12	1	. . .	. . .	. . .	. . .	14
Mixed gram-positive	. . .	. . .	1	. . .	. . .	. . .	. . .	. . .	1
Yeast/fungus	. . .	. . .	. . .	. . .	1	. . .	. . .	. . .	1
H influenzae	. . .	. . .	4	. . .	. . .	. . .	. . .	. . .	4
S marcescens	. . .	1	. . .	. . .	. . .	. . .	. . .	. . .	1
Enterococcus	. . .	2	. . .	. . .	. . .	. . .	. . .	. . .	2
Acinetobacter sp	. . .	. . .	1	. . .	. . .	. . .	. . .	. . .	1
β-hemolytic streptococci	. . .	. . .	. . .	. . .	. . .	1	. . .	. . .	1
Total[a]	6	9	27	2	1	2	5	1	221

[a] There were 168 patients without postoperative infection.

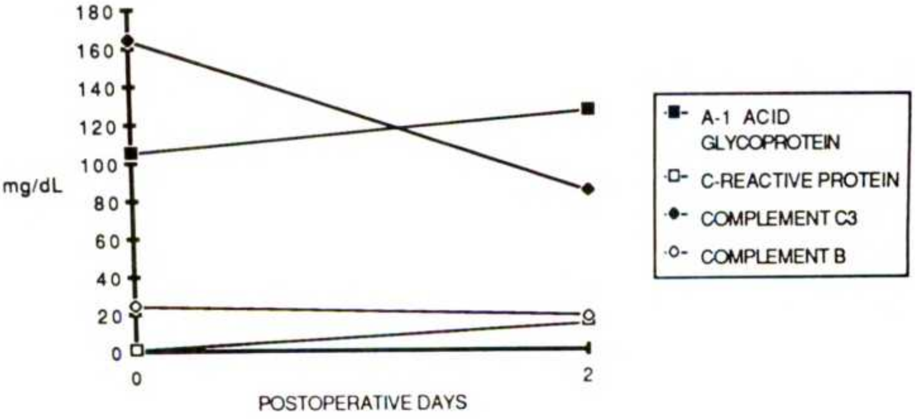
One grade 3 infection grew *Staphylococcus aureus* and the other, mixed gram-positive and gram-negative organisms. The single grade 4 infection was due to *Staphylococcus epidermidis*. There were no concomitant infections in these 6 patients ($p < 0.00001$). Grade 2 infections required only local wound care, whereas grade 3 infections needed extensive debridement. The single grade 4 infection required rotation of myocutaneous flaps for coverage. All 6 patients were eventually discharged. The development of sternal infection extended the mean postoperative hospitalization from 13.8 to 27.3 days ($p < 0.00001$).

Nutritional Status and Acute-Phase Response

Two hundred three patients had quantification of preoperative retinol binding protein, prealbumin, and transferrin. Two hundred one likewise had quantification of complement B, complement C_3, C-reactive protein, and α_1-acid glycoprotein on both the preoperative day and postoperative day 2 (Fig 1). The three nutritional proteins as well as the acute phase proteins were significantly interrelated, as determined by the Pearson product-moment correlation (two-tailed). However, there was no significant relationship between the preoperative concentrations of the three nutritional proteins and the preoperative and postoperative values of the acute phase reactants, with the exception of preoperative C-reactive protein and prealbumin ($p < 0.00001$), and preoperative complement C_3 and transferrin ($p = 0.033$).

One hundred ninety-three (97.5%) of those with well-healed wounds had prealbumin levels within the normal range as did all 5 within this subset in whom sternal infections developed (Figs 2, 3). Although only 39 patients

Fig 1. Time course of the individual acute phase proteins.



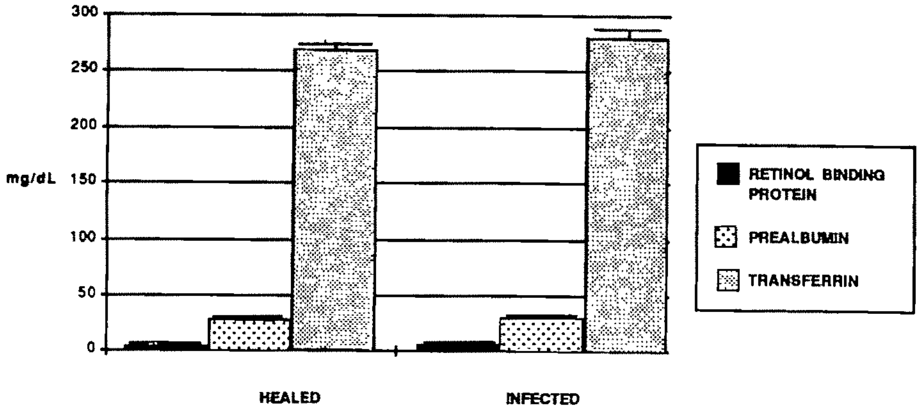


Fig 2. The mean nutritional protein values (± standard error of the mean) of healed patients compared with those who became infected.

(19.7%) with well-healed wounds had abnormal levels of retinol binding protein, 3 of the 5 (60.0%) of those in whom sternal wound complications developed had levels either above or below normal. This did not reach significance by χ^2 analysis. When analyzed as continuous variables, no predictive relationship could be found between these nutritional proteins and the development of sternal infection or other postoperative infections.

Preoperative values of complement C_3 were elevated in 80.0% of those in whom sternal infections developed but only in 30.6% of those (n = 60) with well-healed wounds. Elevated postoperative levels of α_1-acid glycoprotein occurred in 80.0% of those in whom sternal wound complications developed but only in 28.6% of those (n = 56) with well-healed sternal wounds. Again, these trends did not reach significance by χ^2 analysis. Preoperative α_1-acid glycoprotein levels, however, were normal in 82.1% of those with well-healed wounds (n = 161) as well as in 80.0% of those with sternal wound complications. Preoperative complement B levels were normal in 171 patients (87.2%) with well-healed wounds as well as in 80.0% of those in whom sternal wound complications developed. When analyzed as continuous variables, no predictive relationship could be found between the preoperative or postoperative concentrations or the percent change of the acute phase reactants and the development of median sternotomy infection.

Delayed Hypersensitivity

One hundred sixty patients completed evaluation of delayed hypersensitivity response. In the remainder, either they had refused placement of the full set of tests or the tests were not properly recorded. There were no statistical differences between the characteristics of this subset and the group as a whole. There was no correlation between delayed hypersensitivity response and age, sex, weight, diabetes, the use of preoperative steroids, preoperative or postoperative hospitalization, preoperative or postopera-

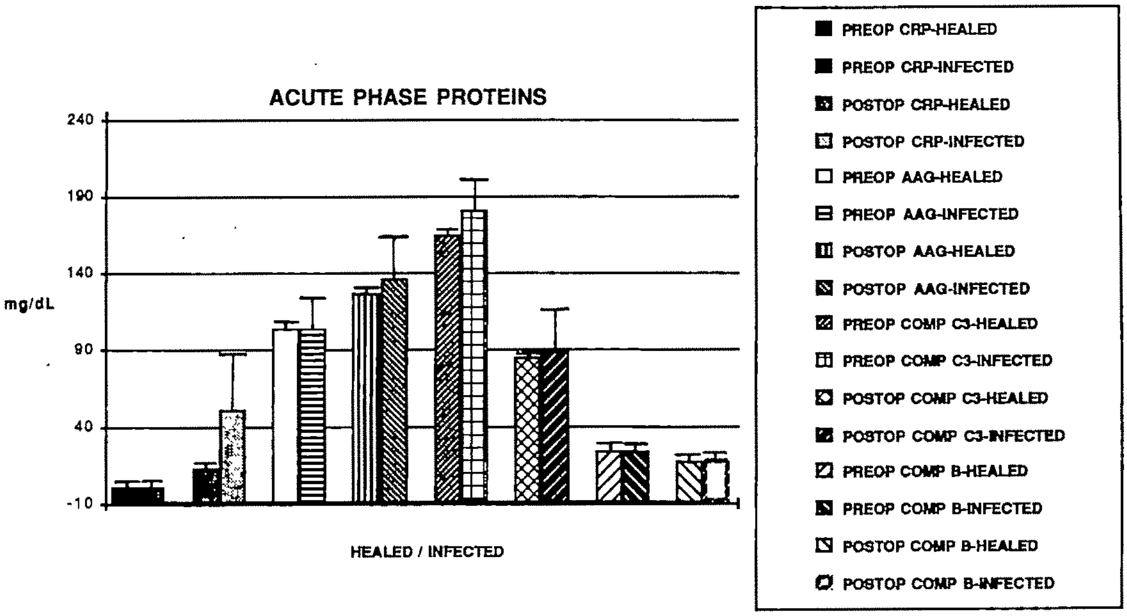


Fig 3. The mean preoperative and postoperative acute phase protein values (± standard error of the mean) of healed patients compared with those who became infected. (AAG = α_1 acid glycoprotein, COMP B = complement B; COMP C3 = complement C_3; CRP = C-reactive protein.)

Table 5. Comparative Delayed Hypersensitivisty Testing

Test	Percent Reactive[a]	
	Healed	Infected
Mumps	12.8 ± 2.8	0.6 ± 0.6
Histoplasma	14.1 ± 0.03	1.3 ± 0.6
Candida	11.5 ± 0.03	0.0 ± 0.0

[a] Values are given as mean ± standard error of the mean.

tive intensive care unit stay, preoperative or postoperative infection or site of infection, cardiopulmonary bypass or aortic cross-clamp time, inotropic support, day of postoperative nutrition, or transfusion requirement.

Of the 160 patients, 117 were anergic (73.1%), 27 were relatively anergic (16.9%), and 16 (10.0%) were normally reactive to either two (n = 12) or three (n = 4) antigens. Twenty-one patients (13.1%) reacted to mumps, 24 patients (15.0%) to *Histoplasma*, and 18 patients (11.3%) to *Candida*. Four patients in this subset died (2.5%) and all were anergic. Four sternal wound infections developed in this subset for a rate of 2.5%. There were two grade 2 infections (one grew *Enterobacter aerogenes*, the other failed to grow in culture), one grade 3 infection (*Staphylococcus aureus*), and one grade 4 infection (*Staphylococcus epidermidis*). Of the 4 patients with sternal complications, 1 was reactive to mumps and *Histoplasma*, 1 to *Histoplasma* alone, and 2 were anergic. There was no statistical correlation between reactivity as a whole and the development of sternal infection. Similarly, comparison between individual antigens and risk of wound infection failed to show a significant relationship (Table 5).

There was no statistical correlation between the nutritional proteins and the delayed hypersensitivity response. Statistically significant relationships between antigen response and the preoperative and postoperative values of the acute phase proteins were noted. A positive reaction to *Histoplasma* correlated ($p = 0.045$) with the preoperative value of complement C_3, and a positive response to *Candida* correlated ($p = 0.035$) with the postoperative value of C-reactive protein. Similarly, a positive skin reaction to mumps antigen corresponded to both the postoperative value of complement C_3 ($p = 0.028$) and the postoperative level of α_1-acid glycoprotein ($p = 0.017$). However, we could find no predictive value in these relationships.

General Clinical Factors

There was no statistical relationship between the development of sternal infection and sex, existence of preoperative infection, length of preoperative hospitalization, diabetes mellitus, ASA class, left ventricular ejection fraction, the use of preoperative steroids, pack-years smoked, room air oxygen tension, operation performed, cardiopulmonary bypass time, reoperation for postoperative hemorrhage, inotropic support, afterload reduction, deprivation of postoperative nutrition, treatment of pericarditis, the individual surgeon, or the type of wound closure.

Patients in whom sternal infections developed were younger (mean age, 57.5 ± 5.2 years; $p < 0.00001$) and significantly heavier (mean weight, 94.8 ± 11.2 kg; $p = 0.00009$). Of the 4 patients requiring closed-chest cardiac compression, grade 2 sternal infections developed in 2 ($p = 0.005$).

Sternal infections developed in 3 (7.7%) of the 39 patients residing in the intensive care unit preoperatively ($p < 0.00001$). Three of the 6 patients in whom sternal infection developed experienced significant postoperative hemorrhage ($p = 0.026$). All three underwent reexploration and received multiple transfusions, but reexploration and transfusion requirement per se did not correlate with the development of sternal sepsis.

Three (7.7%) of the 39 patients undergoing repeat median sternotomy accounted for one half of the six sternal infections ($p = 0.013$). All patients in whom sternal complications developed received internal mammary artery grafts, and the single grade 4 infection occurred with bilateral internal mammary artery harvesting ($p = 0.00006$). However, the operation performed did not correlate significantly with the development of sternal complications.

All sternal wound complications occurred in those closed with a combination of staples and sutures or sutures alone. No sternal infections occurred in the group closed with a subcuticular technique. As expected, there was a significant correlation between the surgeon and type of closure used; however, neither surgeon nor type of wound closure correlated with sternal infection.

Statistical Model

All variables were entered into a stepwise logistic regression analysis with a cut-off point set at 0.5 to formulate a model capable of predicting the individual patient's risk of development of sternal infection. The final logistic model for the predicted risk (%) of sternal wound infection is: PREDSWC = exp(EQ)/1 + exp(EQ); where EQ = (0.38 × age) + (0.24 × weight) + (5.42 × preop ICU) + (4.39 × redo) + (7.14 × IMA) + (4.49 × hemorrhage) + (8.81 × arrest) − 62.72, and preop ICU (intensive care unit), redo, hemorrhage, and arrest are defined as yes (1) or no (0), IMA (internal mammary artery) is defined as 0, 1, or 2, age is in years, and weight is in kilograms. The corrected p values for the above variables are: age, $p = 0.0097$; weight, $p < 0.001$; preop ICU, $p = 0.011$; redo, $p = 0.02$; IMA, $p = 0.0021$; hemorrhage, $p = 0.043$, and arrest, $p = 0.0018$. The sensitivity of this model is 83.3% (5/6) and the specificity is 99.5% (214/215) for this population of patients.

Comment

Nutritional Status

Abel's group [10] looked at nine nutritional parameters including percent ideal weight, triceps skin-fold thickness, mid-arm muscle circumference, percent body fat, serum albumin, serum transferrin, total lymphocyte count, delayed hypersensitivity, and myocardial glycogen concentration. They concluded that this form of nutritional assessment is of no value in guiding the nutritional management of individual patients, although as a group statistical correlations can be made between morbidity and mortality and individual parameters.

Rich and associates [11] recently published results of cardiac operations in patients 75 to 90 years old with serum albumin levels of less than 35 mg/L (3.5 mg/dL) and noted an associated 1.8 relative risk of postoperative infection as well as a 2.25-fold increased hospitalization. Albumin might not be an essential element of wound healing or tissue repair [12], as there are reports of an analbuminemic patient and rats without impairment of growth or wound healing.

In a comparative study of four plasma proteins in malnutrition, albumin had a low sensitivity, transferrin an intermediate sensitivity, and the thyroxin binding prealbumin-retinol binding protein complex the highest sensitivity to alterations of nutritional status [13]. Because the half-life of transferrin is shorter (4 to 8 days) than that of albumin (21 to 28 days), and because it equilibrates rapidly owing to a relatively small extravascular store, it is thought to be more accurate than albumin in assessing protein status [10]. However, the synthesis of transferrin seems to be controlled by contradictory feedback according to protein and iron deficiency, which partly invalidates this test as a reliable index for estimating protein depletion.

Although albumin and transferrin fail to respond to short-term (5 to 7 days) protein or energy restriction, prealbumin and retinol binding protein are very sensitive and respond rapidly to refeeding [6]. The components of the thyroxin binding prealbumin-retinol binding protein complex respond together and in a parallel direction to protein deficiency, apparently related to their rapid turnover rate and unusual richness in tryptophan [13]. Prealbumin and retinol binding protein can be used to detect subclinical malnutrition and monitor the effectiveness of dietary treatment [6, 13]. In the current study, the majority of patients when first seen had normal preoperative nutritional status, and there was no correlation between it and the development of sternal infection.

Acute Phase Response

The metabolic and endocrine responses to trauma are well documented [14]. In vitro [15] as well as in vivo [9] studies have confirmed a state of postoperative immunosuppression. Postoperative or posttraumatic immunosuppression has also been implicated in the development of postoperative sepsis [16]. Injury, through interleukin-1 and prostaglandins, stimulates an increased hepatic synthesis of a number of plasma proteins, termed the acute phase response [7, 12]. C-reactive protein and α_1-acid glycoprotein levels follow a specific time course after surgical trauma [7]. C-reactive protein reaches its maximum on postoperative day 2 irrespective of the trauma severity; α_1-acid glycoprotein reaches its peak level on days 3 through 10 after an elective operation.

The acute phase proteins also have an increased turnover in the peripheral tissues in response to stress and injury [12]. C-reactive protein regulates clotting and inhibits thrombin-activated platelet aggregation with the subsequent release of lysosomal enzymes. C-reactive protein might facilitate healing and reduce the chance of wound infection by stimulating the opsonization of microorganisms. α_1-Acid glycoprotein has structural characteristics similar to both the heavy and light chains of immunoglobulin G and has been shown to inhibit adenosine diphosphate and adrenalin-induced platelet aggregation. α_1-Acid glycoprotein affects the spacing of collagen fibers formed from soluble collagen [12]. These orosomucoids might protect healing tissues from autoimmune reactions, as lymphocyte transformation and phagocytosis are inhibited by α_1-acid glycoprotein. The end result of these actions might be to localize clot formation specifically at the wound, mask potentially antigenic foci, and prevent the production of autoantibodies [12]. Lower preoperative α_1-Acid glycoprotein levels in patients in whom sternal infection later develops might indicate a preoperative impairment of immune status [7]. Degradation and consumption of antiproteases (α_1-acid glycoprotein), complement factors, immunoglobulins, and opsonins (C-reactive protein) might make the organism more susceptible to infection. Degradation of α_1-acid glycoprotein by proteases has been described in experimental septic shock [7]. α_1-Acid glycoprotein, α_1-antitrypsin, complement B, and complement C_3 levels were significantly lower in patients who did not survive a serious septic episode [17].

In the study by Miholic and co-workers [7], α_1-acid glycoprotein and C-reactive protein levels were significantly lower on postoperative day 2 in patients in whom deep sternal infections later developed. There was a marked rise in the concentrations when the infection became clinically evident, paralleled by increases in white blood cell count and temperature. In their study, however, only 47.9% of the initial 188 patients were analyzed with a greater than 11% (10/90 patients) sternal infection rate. It was unclear whether the lower concentrations of these proteins were the effects of decreased synthesis, increased consumption, or increased catabolism, indicative of immunosuppression, or the results of infection. In contrast, we found no relationship between the development of sternal infection and the postoperative concentrations, or percent change, of four acute phase reactants. Despite finding elevated postoperative concentrations of α_1-acid glycoprotein in 80.0% of those in whom sternal infections developed compared with 28.6% of those with well-healed wounds, this trend did not reach statistical significance. Furthermore, we were unable to predict which patients are susceptible to sternal infection based on the preoperative concentrations of the acute phase proteins. Preoperative levels of complement C_3 were elevated in 80.0% of those in whom sternal infections developed but only in 30.6% of those with well-healed wounds, which again did not reach statistical significance. This lack of statistical significance might be due the small number of patients with sternal infection (type II error); a larger study might find these proteins to be predictive.

Delayed Hypersensitivity

Delayed hypersensitivity has usually been viewed as a reflection of cell-mediated immunity and is thought to relate to tumor resistance, transplantation immunity, and resistance to fungal, viral, and intracellular bacterial infections [8]. Anergy has been associated with greater morbidity [9, 10], sepsis, and mortality [8] in preoperative, postoperative, and posttraumatic patients. Johnson's group [9] observed that anergic patients had a threefold risk of postoperative complications compared with the normally reactive or relatively anergic groups combined.

Anergy is closely associated with erosion of the body cell mass and expansion of the intracellular supporting structure as reflected in the total exchangeable sodium to potassium ratio, which is a sensitive index of nutrition [9]. Meakins and associates [8] demonstrated that delayed hypersensitivity reactions tested weekly during an illness and recovery can vary from normal to relatively anergic to anergic and back to normal again. Forse's group [18], using multiple isotope dilution, showed malnutrition was present in anergic patients and not in those who reacted normally. There was considerable overlap, with 43% of reactive patients being malnourished and 21% of anergic patients being normally nourished, and they concluded that skin testing does not accurately predict nutritional status. We too could find no relationship between delayed hypersensitivity and nutritional status.

Movement of both neutrophils and lymphocytes is required for the delayed hypersensitivity response [18]. In anergic patients, the number of lymphocytes is reduced by one third and the number of T cells, by almost one half. Meakins and associates [8] noted abnormal stimulated and random neutrophil migration in anergic patients, and in a graded fashion with respect to anergy and relative anergy. There was a correlation between neutrophil chemotaxis and reactivity. When the delayed hypersensitivity response became normal, chemotaxis returned to near-normal levels. There was also a weak but statistically significant correlation between neutrophil chemotaxis, phagocytosis, and bactericidal killing, implying an inability of the anergic patient to respond to infection.

It has been postulated that C-reactive protein is partially responsible for postoperative anergy owing to inhibition of both lymphokine production and the proliferation of certain T-cell lines [12]. It might stimulate immunoglobulin production at the expense of cell-mediated immunity [12]. C-reactive protein binds to 30% to 40% of all peripheral lymphocytes, binding preferentially to T-cell lymphocytes carrying immunoglobulin G-Fc receptors, which in turn have suppressor activity for B cells in vitro [12]. Therefore C-reactive protein, which might promote a transitory beneficial immunosuppression, could possibly persist at such concentrations as to suppress T-cell as well as B-cell function, compromising the host's resistance to infection [12].

Delayed hypersensitivity response failed to correlate with the development of sternal infection in this study. The majority (73.1%) of our patients were anergic. This apparently was not due to nutritional deficiency as the majority of patients had normal preoperative nutritional protein values, and there was no correlation with deprivation of preoperative or postoperative nutrition. Anergy was not related to duration of cardiopulmonary bypass, aortic cross-clamp time, preoperative or postoperative infection, or inotropic support. The clinical significance of the correlation between individual antigens and either the preoperative or postoperative values of individual acute phase proteins is unknown, and we were unable to predict wound complications with these relationships.

It is unclear whether placing the battery several days preoperatively would have resulted in a greater number of normally reactive patients, or whether the anergy is secondary to some as yet unknown factor specific to cardiac surgical patients. It is possible that using less than five recall antigens resulted in our high rate of anergy since this reportedly decreases the specificity of the categories anergy and relative anergy [9]. Anergy batteries include mumps and *Candida* in most studies [8–10, 18]. Others include *Trichophyton*, purified protein derivative of tuberculin, and streptokinase-streptodornase. *Histoplasma* was chosen in this study due to its endemicity in the Ohio River Valley and therefore a high expected rate of reactivity. We similarly chose to omit purified protein derivative of tuberculin owing to its high expected rate of negativity. An estimated 22% of the United States population are infected with *Histoplasma*, compared with an estimated 5% to 10% who react to tuberculin skin tests [19]. Highly endemic areas center in the great river valleys of the eastern central United States, where in some locations, more than 80% of the population is sensitized.

General Clinical Factors

Wound infection rates after cardiac operations range from 1% to 20% with an acceptable rate of 1% to 3% [1, 3–5]. Our 2.7% infection rate was within the accepted range and would have been lower (1.8%) if the two culture-negative grade 2 sternal wounds had been omitted. Sternal wound infection is caused by contamination of the operative field or by hematogenous spread, which occurs intraoperatively or postoperatively [5]. The contaminating organisms derive from operating room personnel, equipment, the patient, contamination of the extracorporeal circuit, intravenous infusions, blood transfusions, invasive monitoring lines, urinary catheters, and pulmonary infections [2, 3, 5]. As many as 71% of perioperative culture sites are positive [5].

Factors that are not associated with the development of sternal infection include age, sex, weight, diabetes mellitus, preoperative functional class, preoperative cardiac index, intraaortic balloon counterpulsation, emergency operation, total protein on postoperative day 2, and the surgeon's professional status [3, 5, 7]. Our data are in agreement with two exceptions. We noted significant relationships between age and body weight and the risk of sternal infection. Concurrent infection such as in the leg incision, respiratory tract, or urinary tract does not predispose to sternal wound infection [5]. No concurrent infection was documented in our patients in whom sternal infection developed. No significant relationship could be documented between the surgeon, other postoperative infection sites or the organism involved, aortic cross-clamp time, or afterload reduction and the development of sternal infection.

Cardiopulmonary bypass and concomitant hypothermia have complex effects on blood and tissue oxygenation including major changes in the hematocrit, hemoglobin-oxygen dissociation curve, arterial oxygen saturation, and arterial pulsatile flow both during and after operation [2]. Measurements of tissue oxygen content have shown that patients undergoing open heart operations have a decreased oxygen delivery to the wound for a given arterial concentration. Cardiopulmonary bypass also produces complex immunologic effects, as cellular and humoral immunity are impaired [2].

The implication of prolonged operating and bypass times, number of grafts, and excessive bleeding as risk factors for wound complications is controversial [2, 4, 5]. Reexploration for postoperative bleeding has also been implicated as a major risk factor [3–5]. Blood transfusion might alter lymphocyte subpopulations [4, 20], probably owing to concomitant leukocyte transfusion. Suppressor lymphocytes have been found in transfused blood that specifically inhibit cell-mediated alloimmune response [21]. Transfusion might also induce formation of antibodies that inhibit Fc receptors of B lymphocytes, resulting in suppression of host immunity [21]. Transfusion-induced changes in α_2-macroglobulin, a particular protease inhibitor important in fetal protection from maternal immunity, might also affect host immune competence [18]. In this study, postoperative hemorrhage appeared to be a major risk factor, although transfusion requirement and reexploration for postoperative bleeding did not. This was probably due to the large proportion of patients (75.1%) requiring transfusion. Cardiopulmonary bypass time did not correlate with sternal complications although repeat median sternotomy, which is associated with long bypass times, did.

Inotropic agents used to increase central perfusion to the brain, kidneys, and myocardium are likely to decrease both peripheral and wound perfusion [2]. Peripheral and wound perfusion are also compromised by high levels of vasoactive substances such as vasopressin and catecholamines [2]. In the current study, there was no correlation between inotropic support or afterload reduction, both signs of poor perfusion, and the development of sternal infection.

Sternal disruption caused by external cardiac massage, used in early postoperative cardiac arrests, has been associated with a twofold to fourfold risk of wound infection [3–5]. We encountered a 50% sternal infection rate in those patients undergoing closed-chest massage.

The incidence of superficial wound infection after median sternotomy is no greater than after other thoracotomy incisions [5]. After coronary artery bypass grafting, the incidence has ranged from 1.2% to 2.5% and might be even greater when the internal mammary artery is used [4, 5]. Operations for congenital heart disease have a lower incidence of infection, whereas valve replacement falls between [5]. Double-valve replacement or coronary artery bypass with valve replacement has been shown to incur a higher infection rate [3–5], as has triple-valve replacement [3]. We found no correlation between the operation received and the development of sternal infection in this prospective study.

Whether use of the internal mammary artery leads to an increase in sternal complications is controversial. When both internal mammary arteries are used the incidence of sternotomy sepsis might be as high as 8% [3–5, 22]. In this study, there was a significant correlation between the number of internal mammary artery grafts used and the development of sternal infection. An 11% infection rate occurred with bilateral internal mammary grafting.

A preoperative intensive care unit stay was significantly correlated with the development of later sternotomy infection. Whether this simply represents a sicker group of patients with poorer perfusion is doubtful, as both ASA class and left ventricular ejection fraction did not correlate. Although there was a strong correlation between preoperative intensive care unit stay and postoperative infection ($p = 0.00001$), there was no correlation between the occurrence, site, or organism involved preoperatively compared with postoperatively.

Nelson and Dries [1] demonstrated that postoperative sternotomy infection doubles the postoperative stay as well as significantly increases the hospital expense. We too found a significant lengthening of the mean postoperative hospitalization by nearly 13.5 days with the development of sternal wound infection. We did not evaluate the impact of sternal infection on the cost of hospitalization.

Statistical Model

The model presented in this paper is based on stepwise logistic regression. By definition, each predictor variable in the model has independent significance, as is verified by significant p values for each variable. Because four variables are defined as either yes (1) or no (0), all variables are not needed to use the model. The high sensitivity (83.3%) and specificity (99.5%) of this predictive model should be viewed cautiously, as they are based on the data from which the model was derived. Verification against another large group of patients is needed.

Summary

In a prospective study, clinical variables, nutritional status, acute phase response, and cell-mediated immunity were examined for predictive roles in the development of median sternotomy infection. Factors associated with postoperative sternal infection seem to be related to the wound itself. Obesity with a relatively low perfusion of adipose tissue, changes in local circulation that occur with prior median sternotomy and age, transfusion-related immunosuppression as might occur with postoperative hemorrhage, and the disruptive forces that occur with resuscitative measures predispose to the development of sternal infection. Delayed hypersensitivity was not predictive of those in whom sternal infection will develop. A larger study might prove elevated postoperative concentrations of α_1-acid glycoprotein and particularly preoperative levels of complement C_3 to be predictive. We conclude that particular attention to hemostasis, maintenance of hemodynamic stability, preoperative weight reduction, and avoidance of internal mammary grafting in elderly, overweight, and preoperatively unstable patients might decrease the risk of sternal wound infection.

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Pleuropulmonary Morbidity: Internal Thoracic Artery Versus Saphenous Vein Graft

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Although use of the internal thoracic artery (ITA) for coronary artery bypass grafting results in superior graft patency and improved patient survival, our initial clinical observations suggested an increased incidence of pleuropulmonary morbidity with its use. One hundred consecutive patients with left ITA grafts were studied prospectively and compared with a consecutive retrospective group of 100 patients undergoing coronary artery bypass grafting with saphenous vein grafts only. Preoperative, postoperative day (POD) 2, POD 6, and postoperative week 8 chest roentgenograms were analyzed for atelectasis and effusion. Postoperative left lower lobe atelectasis was common in both groups on both POD 2 (saphenous vein, 43%, versus ITA, 53%; not significant) and POD 6 (saphenous vein, 40%, versus ITA, 41%; not significant). There was a significantly higher incidence of pleural effusion on POD 6 in the ITA group (84% versus 47%; $p < 0.05$) but most of these were small. There was more chest tube drainage (1,413 versus 1,028 mL; $p < 0.01$) and a greater need for secondary thoracostomy or thoracentesis (4% versus 0%) in the ITA group. The left pleural space was opened in 67 of the 100 ITA patients but pleurotomy did not appear to influence postoperative morbidity. We conclude that use of the internal thoracic artery for coronary artery bypass grafting results in a small but significant increase in pleuropulmonary morbidity.

(Ann Thorac Surg 1990;50:959–64)

The internal thoracic artery (ITA) has become the conduit of choice for coronary artery bypass grafting (CABG) because of its superior patency rate. Clinical studies have demonstrated that myocardial revascularization with the ITA results in improved relief of symptoms, greater freedom from subsequent myocardial infarction and need for reoperation, and increased long-term survival [1, 2]. Pleuropulmonary complications are relatively frequent after CABG [3]. Postoperative patients commonly have radiologic evidence of pulmonary atelectasis and may demonstrate impaired gas exchange with a widened alveolar-arterial oxygen gradient. A number of factors appear to contribute to these abnormalities including general anesthesia, sternotomy, pleurotomy, cardiopulmonary bypass, and the use of topical hypothermia, which can result in phrenic nerve injury and diaphragmatic dysfunction [3–5]. Use of the ITA requires careful dissection of the vascular pedicle from the posterior aspect of the chest wall and the ipsilateral pleural space is frequently unavoidably opened during the dissection. Some surgeons advocate routine incision of the pleura so as to minimize the risk of tension on the ITA pedicle and distal anastomosis [6]. The procedure of ITA harvesting would appear to increase the potential for pleuropulmonary complications and our own initial clinical experience with ITA grafting did suggest an increase in the incidence of postoperative atelectasis and effusion. This study was accordingly designed to investigate that clinical impression by comparing the incidence of postoperative pleuropulmonary morbidity in patients undergoing CABG with and without the use of the ITA. The relationship of pleurotomy to subsequent pleuropulmonary abnormalities in ITA patients was also examined.

Material and Methods

Clinical Data

One hundred consecutive patients undergoing CABG using saphenous vein grafts (SVGs) only were compared with 100 consecutive patients undergoing CABG using the left ITA, either alone or in combination with SVG. The SVG patients underwent operation over a 9-month period starting in January 1984. The ITA patients were studied prospectively from September 1987 until March 1988. Patients undergoing redo coronary artery bypass operations or patients requiring prolonged postoperative ventilation (>48 hours) were excluded, as were patients requiring insertion of a chest tube in the right pleural space.

Dissection of the ITA pedicle was completed before heparinization and cannulation. Entry into the pleural space was avoided where possible. Cardiopulmonary bypass was instituted with a bubble oxygenator (BOS-10, Bentley Ltd) in the SVG group and with a membrane oxygenator (CML-50-100, Cobe Laboratories) in the ITA

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group as our institution switched to the membrane oxygenator in 1986. Apart from the difference in oxygenator and use of the ITA, the surgical approach and conduct of cardiopulmonary bypass were the same in the two groups. Moderate hypothermia (28° to 30°C) and cold (4°C) crystalloid cardioplegic solution were used for myocardial preservation. Myocardial hypothermia was further enhanced by the use of a cooling jacket (Cobe Laboratories) around the heart. An intrapericardial insulating pad (Shiley Canada) was used to prevent hypothermic damage to the phrenic nerve. Distal anastomoses were carried out during a single period of aortic cross-clamping, whereas the proximal anastomoses were completed using a tangential clamp on the ascending aorta. If the pleural space had been entered, care was taken to evacuate any accumulated blood before chest tube insertion and sternal closure.

Data collected for each patient included age, sex, number of distal anastomoses, ischemic time, cardiopulmonary bypass time, pleural space entry, duration of postoperative ventilation, chest tube drainage, and length of hospital stay after operation. Chest tubes were routinely removed when total drainage was less than 100 mL over the previous 12 hours. Patients requiring either thoracentesis or secondary chest tube insertion up until the 8-week postoperative follow-up visit were identified.

Radiologic Assessment

Preoperative, postoperative day (POD) 2 and POD 6 chest roentgenograms were reviewed by a single radiologist (M.L.). Radiographs were randomized to permit blinding as to postoperative duration but not patient group as this was precluded by the use of metallic clips for ITA mobilization. Follow-up postoperative week 8 chest roentgenograms in the ITA patients were also reviewed; these were not available for analysis in the SVG group. Preoperative, POD 6, and postoperative week 8 films included standard posteroanterior and lateral chest roentgenograms, as well as a left lateral decubitus film on postoperative week 8 in the ITA group. Postoperative day 2 films were single anteroposterior views taken while the patients were in the surgical intensive care unit. All films were assessed for left-sided parenchymal changes and left pleural effusion using a graded scale from 0 to 3 (Appendix 1).

Statistical Analysis

Data are reported as the mean ± standard error of the mean. Patient variables were compared using the unpaired Student's t test; χ^2 analysis was used to interpret results of the chest roentgenogram scoring. A p value less than 0.05 was considered statistically significant.

Results

Clinical Observations

There were no significant differences between the two groups with respect to percent male, number of distal anastomoses, ischemic time, or cardiopulmonary bypass time (Table 1). The ITA patients were slightly older, with a mean age of 58.7 years compared with 54.7 years for the SVG group ($p < 0.01$). The left pleural space was opened in 67 of 100 ITA patients and 7 of 100 SVG patients. Postoperative ventilation time averaged 25.7 hours in the SVG group and 21.9 hours in the ITA group ($p < 0.01$). Total chest tube drainage was significantly greater in the ITA group (ITA, 1,286 ± 56 mL, versus SVG, 1,089 ± 48 mL; $p < 0.01$). One patient in the ITA group was returned to the operating room several hours postoperatively for cardiac tamponade, and a bleeding site on the ITA pedicle was identified and controlled. A superficial wound infection developed in 1 patient in the ITA group. There were no cases of sternal wound infection in either group, nor did any patient require treatment for bronchopneumonia. Mean postoperative hospitalization was 9.9 ± 0.3 days in the ITA group and 10.9 ± 0.3 days in the SVG group ($p < 0.05$). There were no deaths in either consecutive series of patients.

Table 1. Baseline Variables for SVG and ITA groups.[a]

Variable	SVG	ITA	p Value
Age (y)	54.7 ± 0.8	58.7 ± 0.9	<0.01
Percent male	84	91	NS
Distal anastomoses	2.9 ±0.1	2.9 ± 0.1	NS
Ischemic time (min)	43.3 ± 1.3	47.2 ± 1.6	NS
Cardiopulmonary bypass time (min)	88.1 ± 2.4	92.3 ± 3.0	NS

[a] Values are mean ± standard error.

ITA = internal thoracic artery; NS = not significant; SVG = saphenous vein graft.

Radiologic Assessment

There was no radiologic evidence of atelectasis or pleural effusion in any patient preoperatively. Left lower lobe atelectasis was a common finding in both study groups on both POD 2 (SVG, 43%, versus ITA, 53%; not significant

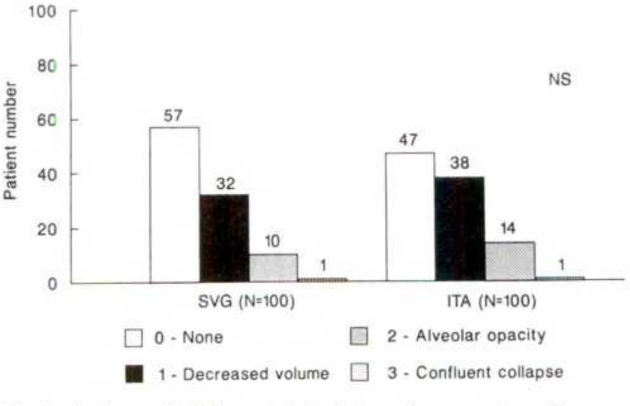

Fig 1. Incidence of left lung atelectasis in saphenous vein graft (SVG) only and internal thoracic artery (ITA) patient groups on postoperative day 2. (NS = not significant.)

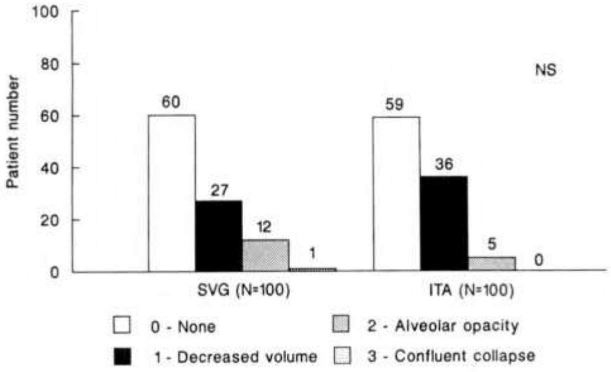

Fig 2. Incidence of left lung atelectasis in saphenous vein graft (SVG) only and internal thoracic artery (ITA) patient groups on postoperative day 6. (NS = not significant.)

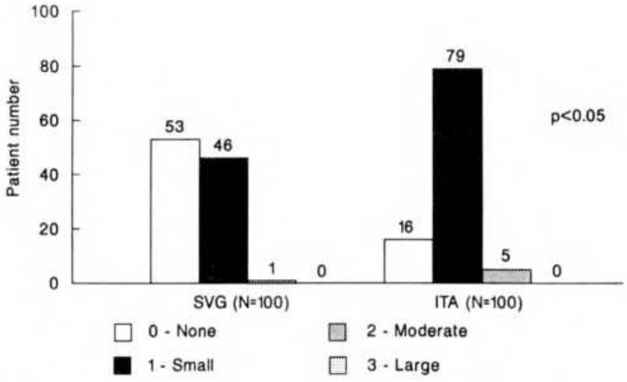

Fig 4. Incidence of left pleural effusion in saphenous vein graft (SVG) only and internal thoracic artery (ITA) patient groups on postoperative day 6.

[NS]) (Fig 1) and POD 6 (SVG, 40%, versus ITA, 41%; NS) (Fig 2). There were no significant differences between the two groups with regard to effusion on POD 2 (Fig 3). There was, however, a significantly greater incidence of effusion in the ITA patients on POD 6 (Fig 4). A total of 84 patients in the ITA group had effusions (79 small, 5 moderate) as opposed to only 47 patients in the SVG group (46 small, 1 moderate) ($p < 0.05$).

Pleurotomy Versus No Pleurotomy

The left pleural space was entered in 67 of the 100 ITA patients. Total chest tube drainage was significantly greater in these patients as was the duration of postoperative ventilation (Table 2). Effusions were more common in patients without pleurotomy on POD 2 but there was no difference between the two groups by POD 6 (Table 3). The incidence of parenchymal changes consistent with atelectasis was similar in both groups on POD 2 but appeared slightly increased in the patients without pleurotomy on POD 6 (see Table 3). Follow-up at 8 weeks after bypass was complete in 76 of the original 100 ITA patients (pleurotomy, 55; no pleurotomy, 21). The large majority of patients were free of atelectasis (pleurotomy, 94.5%, versus no pleurotomy, 95.2%; NS). Small pleural effusions were present in 9.1% of the pleurotomy patients and 14.3% of the patients without pleurotomy (NS). Clinically significant diaphragmatic elevation was not seen in any patient.

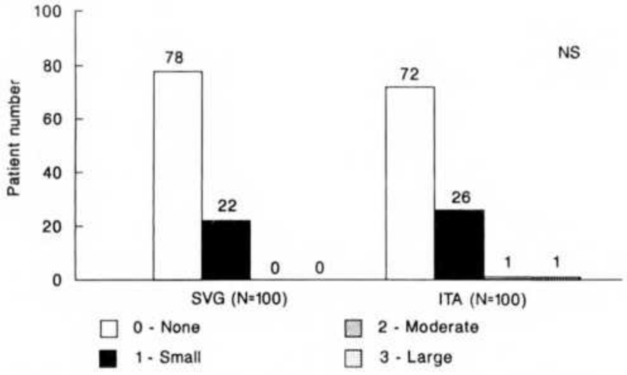

Fig 3. Incidence of left pleural effusion in saphenous vein graft (SVG) only and internal thoracic artery (ITA) patient groups on postoperative day 2. (NS = not significant.)

Secondary Procedures

Four patients in the ITA group required subsequent drainage procedures for postoperative pleural effusion. In 1 woman a large left pleural effusion developed over the initial 48 hours after operation; it was successfully treated with chest tube drainage. Two male patients required thoracentesis for large residual loculated effusions. One patient was still in the hospital (POD 7) whereas the other was identified at the 8-week follow-up visit. Both patients were asymptomatic. Dyspnea developed in the final pa-

Table 2. Internal Thoracic Artery Patients With and Without Pleurotomy[a]

Variable	Pleurotomy	No Pleurotomy	*p* Value
No. of Patients	67	33	<0.01
Age (y)	58.8 ± 1.1	58.4 ± 1.4	NS
Distal anastomoses	2.9 ± 0.2	2.9 ± 0.2	NS
Ischemic time (min)	46.8 ± 2.0	48.1 ± 2.7	NS
Cardiopulmonary bypass time (min)	92.1 ± 3.7	92.6 ± 4.9	NS
Total chest tube drainage (mL)	1,413 ± 72	1,028 ± 66	<0.01
Ventilation time (h)	22.9 ± 0.8	19.8 ± 0.8	<0.01
Postoperative hospitalization (d)	10.1 ± 0.4	9.5 ± 0.5	NS

[a] Values are mean ± standard error.

NS = not significant.

Table 3. Incidence of Early Pleuropulmonary Morbidity in Internal Thoracic Artery Patients With and Without Pleurotomy[a]

POD	Grade[b]					p Value
	0	1	2	3	1 + 2 + 3	
Atelectasis						
POD 2						
P	49.3	41.8	9.0	0	50.7	NS
NP	42.2	30.3	24.2	3.0	57.6	
POD 6						
P	67.2	31.3	1.5	0	32.8	<0.05
NP	42.2	45.5	12.1	0	57.6	
Pleural Effusion						
POD 2						
P	82.1	17.9	0	0	17.9	<0.05
NP	51.5	42.4	3.0	3.0	48.5	
POD 6						
P	14.9	82.1	3.0	0	85.1	NS
NP	18.2	72.7	9.1	0	81.0	

[a] Values are mean percentages ± standard error. [b] See Appendix 1 for definition of grading scale used for radiologic assessment.

NP = no pleurotomy; NS = not significant; P = pleurotomy; POD = postoperative day.

tient 2 weeks after discharge from the hospital; this patient was readmitted for chest tube drainage of a large recurrent left pleural effusion. Pleural space entry at the time of operation was recognized in only 1 of these 4 patients (the man requiring thoracentesis at 8 weeks). No patient in the SVG group required either thoracentesis or chest tube insertion postoperatively.

Comment

Varying degrees of pulmonary dysfunction are common after CABG. Postoperative pulmonary atelectasis results in ventilation-perfusion mismatch and a widened alveolar-arterial oxygen difference. A number of factors appear contributory in addition to the patient's preoperative cardiopulmonary and postoperative hemodynamic status. General anesthesia itself results in a significant decrease in functional residual capacity and various inhalational agents are known to inhibit hypoxic pulmonary vasoconstriction [7]. It has been demonstrated that sternotomy for CABG results in a significant decrease in postoperative vital capacity, forced expiratory volume, and other indices of pulmonary function [8].

Cardiopulmonary bypass has been shown to result in complement activation [9] and pulmonary leukocyte sequestration [10, 11], and this has been correlated with postoperative pulmonary dysfunction [12]. Pulmonary ischemia during the period of aortic cross-clamping, pulmonary injury secondary to the high potassium concentration in the cardioplegic solution traversing the pulmonary microcirculation, and hypothermia have all been postulated as causes of impaired production or function of surfactant [3, 13]. The use of topical ice slush to enhance myocardial cooling has been clearly demonstrated to result in a higher incidence of elevated hemidiaphragm, presumably secondary to hypothermic phrenic nerve damage [5]. This phrenic nerve dysfunction may be associated with serious postoperative respiratory complications, which may be largely preventable by the use of a pericardial insulating pad [14].

It might be anticipated that use of the ITA for CABG would result in a higher incidence of pleuropulmonary morbidity than that seen with the use of vein grafts only. Mobilization of the arterial pedicle with transection of associated lymphatic channels might be expected to result in subsequent pleural fluid accumulation. The increased chest wall retraction required for exposure and the additional dissection needed to mobilize the ITA pedicle might lead to greater postoperative discomfort with less efficient coughing and pulmonary mechanics. Berrizbeitia and associates [8] demonstrated a significant deterioration in pulmonary function tests in patients after CABG, and this decrease in function was greater in the group in which the ITA was harvested.

It is frequently necessary to enter the adjacent pleural space while mobilizing the ITA pedicle. Furthermore, in some cases it may be advantageous to open the pleural space to afford the shortest course for the ITA and to thus minimize the risk of anastomotic tension. Burgess and colleagues [4] measured postoperative blood gases and calculated pulmonary shunt fractions in 32 patients after elective CABG with and without the ITA. They found that patients who received ITA grafts had significantly higher pulmonary shunt fractions after bypass and significantly lower postoperative arterial oxygen tensions than patients who received SVG only. A subgroup of vein graft only patients in whom the pleural space was opened showed results similar to the ITA group, leading Burgess and colleagues to conclude that it was pleurotomy rather than the use of the ITA per se that led to the increase in pulmonary shunting after bypass. Ghia and Anderson [15] also reported an adverse effect of pleurotomy on pulmonary function before and after cardiopulmonary bypass, although none of their patients received ITA grafts.

In this study we saw no difference in the incidence of atelectasis between the patients who did and did not receive ITA grafts. Approximately half of the patients showed roentgenographic evidence of atelectasis on POD 2, and this incidence decreased to approximately 40% by POD 6. No patient was diagnosed as having postoperative bronchopneumonia. Residual atelectasis was very uncommon at the 8-week follow-up, being present in only 5% of patients.

There was also no difference between the two groups with respect to the incidence of pleural effusion on POD 2. This is not unexpected in view of the fact that pleural drainage tubes would generally still be in place at this time. By POD 6, there was a significantly greater incidence of effusion in the ITA group, but most of these effusions were small. Total chest tube drainage was significantly greater in the ITA group, but the difference of approximately 200 mL was relatively modest.

Somewhat to our surprise, there was no major benefit seen in the one third of ITA patients in whom pleurotomy was avoided. Opening of the pleural space was associated with a slightly longer duration of postoperative ventilation and a significantly greater amount of total chest tube drainage. The latter finding has also been reported by Olearchyk and Magovern [16], and it has been suggested that pleurotomy itself contributes to the production or interferes with the lymphatic clearance of pleural fluid [17]. It is noteworthy that all 4 patients requiring thoracentesis or secondary chest tube insertion were in the ITA group and that 3 of the 4 were in the "no pleurotomy" subgroup. It seems reasonable to assume that delayed accumulation of pleural fluid may occur when small openings made in the pleura at operation are unrecognized but subsequently permit translocation of fluid from the mediastinum to the pleural space. Several similar cases were recently documented and discussed by Kollef and associates [17].

Ideally, this study would have been best carried out in a prospective fashion with patients randomized to either ITA or SVG only groups. The clear superiority of the ITA as a bypass conduit under most circumstances, however, would make such a study ethically questionable at this time. The two groups underwent operation approximately 3½ years apart. Before this, saphenous vein grafts were used almost exclusively, whereas at the time the study was initiated virtually all patients received at least one ITA graft. During the intervening period, younger low-risk patients were receiving ITA grafts while older and higher-risk patients were receiving vein grafts only. Thus, ITA and SVG patients from this time period would clearly not be comparable. It was thus necessary to go back 3½ years from the time the study was initiated to obtain a closely matched control group.

Despite the time interval, the two groups were closely comparable with respect to demographic data (see Table 1) and clinical management, and both groups were composed of consecutive patients coming for CABG. The mean age of the ITA group was 4 years greater than that of the SVG group and reflects the well-recognized trend toward an aging surgical population. The small but significant decreases in postoperative ventilation time and hospital stay in the ITA group were more likely a reflection of changing clinical practice than a true difference in clinical course between the two groups.

In conclusion, use of the ITA for CABG appears to result in a small but statistically significant increase in pleuropulmonary morbidity. This has been documented by a higher incidence of pleural effusion (84% versus 47%, $p < 0.05$), increased chest tube drainage (1,413 versus 1,028 mL; $p < 0.01$), and a greater need for secondary thoracostomy or thoracentesis (4% versus 0%). Despite this small increase in morbidity, continued widespread use of the ITA for CABG appears justified in view of its superior patency rate.

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Appendix 1. Radiologic Assessment

Atelectasis

0 = Normal
1 = Crowded markings, blurred vessels, decreased volume
2 = Alveolar opacities
3 = Homogeneous opacities, confluent collapse

Effusion

0 = None
1 = Small
2 = Moderate
3 = Large

Notice to Readers of *The Annals of Thoracic Surgery*

Dr Richard M. Peters, who has functioned as an Associate Editor in charge of book reviews for the past 11 years, at his request retired from this position at the time of the Interim Meeting of The Society of Thoracic Surgeons in Chicago, September 21, 1990. He will remain as an Editorial Board member. Dr Peters has performed a great service for the readers of *The Annals*, and we are grateful to him.

Dr J. Kent Trinkle has agreed to be the new Associate Editor in charge of book reviews. Any correspondence concerning this section of *The Annals* should be sent directly to him at the address listed below:

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Medical Management of Acute Traumatic Rupture of the Aorta

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Surgical reconstruction is the treatment for acute traumatic aortic rupture and should be accomplished immediately in most patients. In patients in whom concomitant injuries or the development of life-threatening complications preclude safe and successful aortic reconstruction, pharmacological intervention to reduce the risk of free aortic rupture may be considered. Surgical reconstruction can then be more safely performed under controlled elective circumstances.

(Ann Thorac Surg 1990;50:965–7)

Acute traumatic disruption of the thoracic aorta is a life-threatening complication of rapid deceleration injuries. It is commonly accepted that urgent surgical repair is indicated. However, when the aortic injury is initially stable and when the patient has other serious associated injuries that require immediate attention, such as massive intraabdominal hemorrhage or severe central nervous system injury, pharmacological intervention to lower the systolic blood pressure and to reduce the cardiac shear forces may serve as temporary management to decrease the probability of free rupture until surgical repair of the aorta may be carried out more safely.

The use of pharmacological agents in the treatment of aortic dissection was introduced by Wheat and colleagues in 1965 [1]. The concept of pharmacological intervention for management of acute traumatic aortic disruption is not new, but it is not well appreciated and should be reiterated. We have operated on 79 patients with acute traumatic aortic rupture. Because of complicating factors, 3 recent patients were stabilized medically until aortic repair could be more safely performed. Free rupture did not occur in any patient. The following case illustrates this rather unorthodox approach to traumatic aortic rupture.

A 41-year-old man involved in a motor vehicle accident was admitted in severe respiratory distress. Examination showed a flail right hemithorax, right humeral and clavicular fractures, bilateral pubic rami fractures, and a silent and diffusely tender abdomen. Gross blood was obtained on diagnostic peritoneal lavage. Chest roentgenogram demonstrated multiple right-sided rib fractures and a hemopneumothorax, which was treated with tube thoracostomy. Immediately after initial resuscitation, the patient's abdomen was explored. Laparotomy disclosed a linear fracture of the liver and a bulging right hemidiaphragm. A second chest tube was inserted, followed by an anterolateral thoracotomy when a massive air leak persisted. Severely lacerated and contused middle and lower lobe lung injuries were controlled with resection of considerable macerated lung and multiple suture repairs of other individual lacerations.

The patient required increasing ventilatory support during the first 12 hours owing to hypoxemia (oxygen tension of 55 mm Hg on 100% inspired oxygen fraction) and loss of pulmonary compliance. A second chest roentgenogram showed an indistinct aortic knob that was not initially noted and a left-sided pleural effusion that was bloody when drained through tube thoracostomy. An aortogram was obtained, demonstrating complete disruption of the aorta at the isthmus (Fig 1). Owing to the patient's deteriorating respiratory status, it was thought that he would not survive general anesthesia. Using continuous arterial pressure monitoring through a radial artery cannula and pulmonary artery catheterization for frequent measurements of cardiac function, the patient was begun on a regimen of intravenous sodium nitroprusside to reduce the systolic blood pressure and intravenous propranolol hydrochloride to reduce the heart rate and to decrease the slope of the left ventricular ejection curve. His hospital course was complicated by cardiac contusion and dysrhythmias, acute renal failure, staphylococcal pneumonia and empyema, and prolonged ventilatory support. After 6 days, intravenous methyldopa was substituted for the sodium nitroprusside and intravenous administration of propranolol hydrochloride was continued until the patient's clinical condition allowed these drugs to be administered orally. He recovered sufficiently from his injuries to be discharged on the 60th hospital day on a regimen of methyldopa and propranolol hydrochloride while his renal failure slowly resolved.

Three weeks later, a second aortogram showed a false aneurysm at the site of rupture (Fig 2). He underwent an uneventful and easy aortic repair 88 days after the injury using a Dacron interposition graft and partial femoral vein–artery bypass. The aortic ends were approximately 2 cm apart in a small, well-fibrosed false aneurysm. His postoperative course was uneventful, and he was discharged 8 days after repair.

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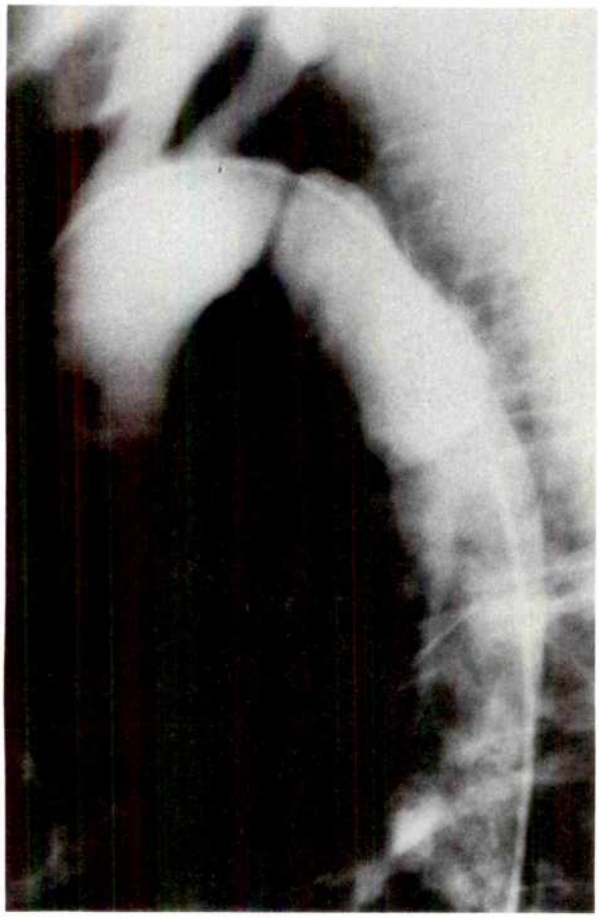

Fig 1. Initial aortogram demonstrating complete transection of the thoracic aorta distal to the left subclavian artery.

Comment

Successful management of patients with traumatic disruption of the thoracic aorta depends on prompt suspicion of the injury based on the mechanism of injury and usually on a widened mediastinum on chest roentgenogram. Early definitive diagnosis requires emergent aortography. Immediate surgical reconstruction of the aorta may then be accomplished in the majority of patients. The operative technique we use has previously been reported [2].

There is a select group of patients with traumatic aortic disruption who are not candidates for immediate reconstruction. This group includes patients with massive intraabdominal bleeding, severe central nervous system injury, critical pulmonary injury, or severe burns to the torso, and those with delayed diagnosis and in whom serious complications that would preclude safe general anesthesia develop. In these patients, the continuity of the aorta is maintained by the adventitia similar to an acute dissecting hematoma of the aorta. Pharmacological intervention in this group of patients was first alluded to by Aronstam and associates [3] as a temporizing maneuver to prevent free rupture of the aorta while awaiting clinical improvement in the patient's condition. Medical therapy is directed at reducing the systolic blood pressure and at decreasing the cardiac ejection velocity. In a review of the English literature in 1985, Svensson and colleagues [4] found 44 cases of traumatic aortic injury treated using this method. Since 1985, 20 additional cases have been reported [5, 6]. In all cases reported, free rupture of the aorta did not occur while the patient was receiving aggressive medical therapy.

We report our success in managing 3 patients with traumatic aortic disruption using an initial regimen consisting of an intravenous vasodilator (sodium nitroprusside) to lower the systolic blood pressure and intravenous β blockade (propranolol hydrochloride) to reduce the cardiac impulse when immediate aortic reconstruction was not feasible.

We recommend that patients with suspected aortic injuries undergo aortography on an emergent basis. Pa-

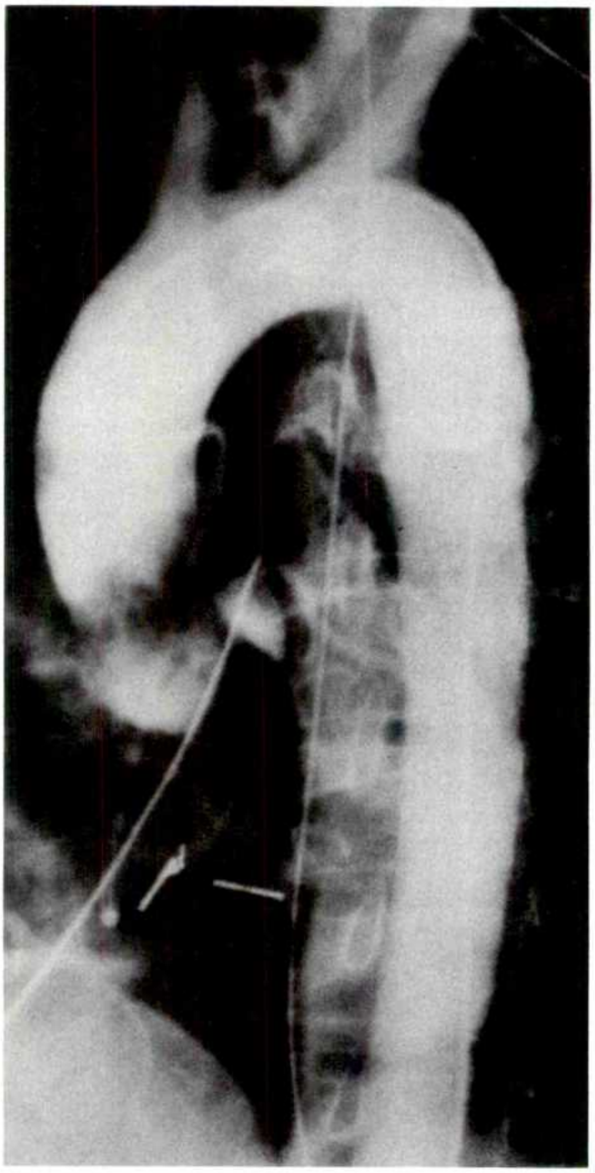

Fig 2. Aortogram approximately 12 weeks after injury demonstrating a false aneurysm of the thoracic aorta at the site of rupture.

tients with severe head injuries should undergo cerebral computed tomographic scanning before undergoing aortography with the blood pressure closely monitored and controlled. Patients with positive diagnostic peritoneal lavage and continuing hypotension should proceed to immediate laparotomy. The patient should be positioned to allow immediate access to the left chest, and the laparotomy should be performed rapidly in a manner compatible with possible systemic heparinization (eg, splenectomy rather than splenorraphy). Once intraabdominal hemorrhage is arrested, attention should be given to maintaining the systolic blood pressure below 110 mm Hg pharmacologically. After abdominal exploration, aortography is performed, and if an aortic injury is found, the patient is returned to the operating suite for aortic reconstruction.

If a patient's condition does not allow for urgent repair of the aorta, pharmacological intervention is continued in an attempt to control blood pressure and to reduce the cardiac impulse. Continuous arterial pressure monitoring and pulmonary artery catheterization to monitor cardiac output and optimize cardiac function and fluid status are imperative in this situation. When the patient is stabilized and is swallowing normally, oral drugs may slowly replace intravenous drugs. When the clinical condition of the patient is optimal, aortic repair can be performed as an elective procedure.

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Surgical Epicardial Ablation of Left Ventricular Pathway Using Sling Exposure

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We report our experience with 43 consecutive patients with left free wall pathways operated on since December 1988 using a modified direct epicardial approach through a medial sternotomy, without the adjunct of normothermic cardiopulmonary bypass. The left atrioventricular sulcus is exposed by dislocating the heart cephalad and to the right using a sling made of a large sponge passed around the ventricle through the transverse sinus. While the arterial pressure is monitored, the heart is positioned to obtain adequate exposure without compromising the ventricular function. The left atrioventricular junction is exposed using a direct approach. The epicardium is incised along the ventricular edge and a plane of dissection is identified and opened using blunt dissection over the ventricular wall. The entire left atrioventricular junction can be exposed. After dissection, electrophysiological testing is repeated to assess accessory pathway conduction. Epicardial cryoablation was used when accessory pathway conduction was not present (42 patients). Transmural cryoablation was used under normothermic cardiopulmonary bypass when accessory pathway conduction persisted after dissection (subendocardial pathway). In all, cardiopulmonary bypass was not used in 41 patients. There was one early relapse that required transmural cryoablation. There were no complications.

(Ann Thorac Surg 1990;50:968–71)

The epicardial approach [1, 2] for ablation of accessory atrioventricular (AV) pathways in patients with problematic Wolff-Parkinson-White syndrome was developed to diminish surgical risk by avoiding adjuncts such as cold cardioplegic cardiac arrest and cardiopulmonary bypass. Right ventricular free wall accessory pathways were ablated without the use of cardiopulmonary bypass [3], as were the vast majority of posterior septal pathways [4–6]. In rare instances, left ventricular free wall pathways were ablated through a left thoracotomy without the support of cardiopulmonary bypass [3]. Because lateral thoracotomy is associated with greater morbidity than median sternotomy [7] left free wall pathways have been approached through a median sternotomy and epicardially ablated under normothermic cardiopulmonary bypass [3].

For editorial comment, see page 866.

Since December 1988, we have used an exposure of the left ventricular region: the sling exposure, which allows surgical ablation of left free wall accessory pathways without the adjunct of cardiopulmonary bypass through a median sternotomy.

Material and Methods

Patients

Our experience comprised 43 consecutive patients with left free wall accessory AV pathways. There were 25 women and 18 men. Their mean age 33 ± 13 years. The mean duration of symptoms was 15 ± 10.6 years, ranging from 2 weeks to 20 years. All patients had palpitations. Nine patients had syncope and 12 patients were cardioverted at least once. Eleven had associated chest pain. One patient had clinically significant associated coronary artery disease.

All patients had preoperative electrophysiological study. Thirty patients had manifest Wolff-Parkinson-White syndrome. The AV reentrant tachycardia mean cycle length was 332 ± 50 ms. The shortest RR interval between two preexcited beats during induced atrial fibrillation was 244 ± 57 ms. Forty-two patients had single left free wall pathways. One patient had multiple pathways with a left free wall pathway associated with an anterior septal pathway.

Surgical Technique

The heart was exposed via an extended median sternotomy in all patients. The skin incision was prolonged into the epigastric region 2 to 4 cm above the umbilicus. The pericardium was opened using a routine vertical incision combined with a transverse incision along the diaphragm. The transverse pericardial incision combined with dissection of the peritoneal sac from the rectus abdominis in the epigastric region allows marked caudal deflection of the inferior pericardial wall. This maneuver produces good exposure of the inferior wall of the heart as well as the obtuse margin (Fig 1).

INTRAOPERATIVE ELECTROPHYSIOLOGICAL TESTING. Epicardial activation mapping was carried out using a hand-held

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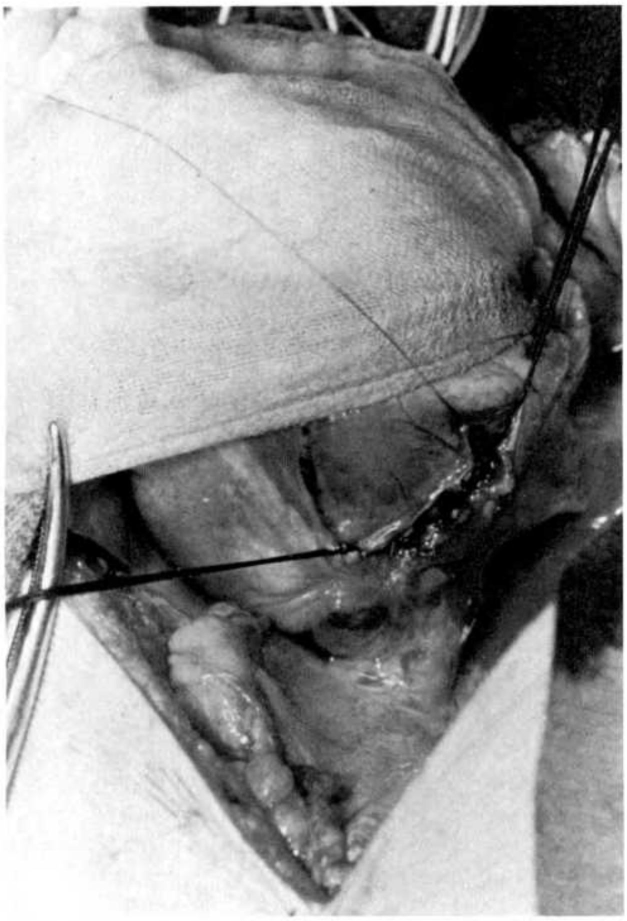

Fig 1. Panoramic operative view depicting the dislocation of the heart by the sling, exposure of the left atrioventricular sulcus, and direct epicardial approach.

roving probe applied at 17 predetermined sites along the atrioventricular sulcus [8]. Ventricular mapping was performed during atrial pacing. Atrial mapping was carried out during reciprocating tachycardia or ventricular mapping. A semicircular multielectrode probe was used along the left ventricular free wall to prevent interruption of accessory pathway conduction associated with dislocation of the heart. There was good correlation between preoperative and intraoperative localization of the accessory pathway in all patients. The left ventricular region was divided into three segments: the mid-left ventricular region (obtuse margin), the left coronary fossa or so-called left anterior septal region, and the left paraseptal region, which is the transitional segment between the mid-left ventricular segment and the posterior septal region.

SLING EXPOSURE. Sling exposure was carried out as follows: (1) The string and adjacent corner of a laparotomy sponge was passed through the transverse sinus from left to right and the string clamped over the sheet covering the right edge of the median sternotomy. (2) A side of the quadrangular sponge was wrapped along the base of the left ventricle and the inferior right ventricle and attached onto the right edge of the pericardial incision using a Kelly clamp. (3) The rest of the sponge was wrapped around the ventricles and folded to construct a sling. (4) The ventricles were dislocated within the sling, the free tip of which was attached onto the right edge sheet when desired exposure of the left ventricular AV sulcus was obtained. During dislocation, arterial pressure and mixed venous blood oxygen saturation were monitored. Adequate dislocation was a compromise between hemodynamic preservation and adequate exposure. As the site of dissection moved around the left AV sulcus, the sling exposure was adjusted accordingly.

Steady hemodynamics were attained before dissection: systolic arterial blood pressure greater than 90 mm Hg and unchanged mixed venous blood oxygen saturation. Progressive hemodynamic deterioration associated with global ventricular hypocontractility was associated with compression of the left main or its bifurcation in the left coronary fossa or the transverse sinus by the laparotomy sponges. The heart dislocation was terminated immediately and the heart quickly recovered. The laparotomy sponge was repositioned and the heart could be again dislocated without complication in all cases.

OPERATIVE THERAPY. The epicardial approach combined exposure and cryoablation of the atrioventricular junction on the beating heart during continuous electrophysiological monitoring. Accessory pathway conduction was assessed before dissection and after exposure of the atrioventricular junction. The majority of left ventricular free wall accessory pathways were epicardial, coursing within the AV sulcus [3]; their conduction was not present after dissection of the AV junction and their ablation was ensured by epicardial cryoablation. When accessory pathway conduction was present after extensive dissection of the AV junction, accessory pathways were classified as subendocardial [9], potentially crossing the AV junction endocardially. Transmural cryoablation requiring the adjunct of cardiopulmonary bypass was used for uniform ablation of subendocardial pathways.

The direct epicardial approach, which requires lesser ventricular dislocation for adequate exposure (Fig 2), was used in place of the en bloc epicardial approach previously described [6]. The AV junction was exposed by direct dissection of the AV fat pad along the left ventricular wall as follows: (1) The epicardium was incised along the left ventricular wall in the inferior segment of the AV sulcus. The dissection was commenced at that site because it is usually free of coronary arteries. (2) A plane of dissection was safely identified over the left ventricular wall. (3) The obtuse marginal vein or its variants was ligated and divided. Generally, the vein ran deep into the fat pad although it appears superficial over the left ventricular wall. Depending on anatomical configuration, division of veins was not always required. (4) Division of the obtuse marginal vein allowed opening of the plane of dissection and adequate exposure to identify and dissect the coronary arteries. According to anatomical configuration, the circumflex coronary artery and its branches were mobilized en bloc onto the left ventricle, or ventricular

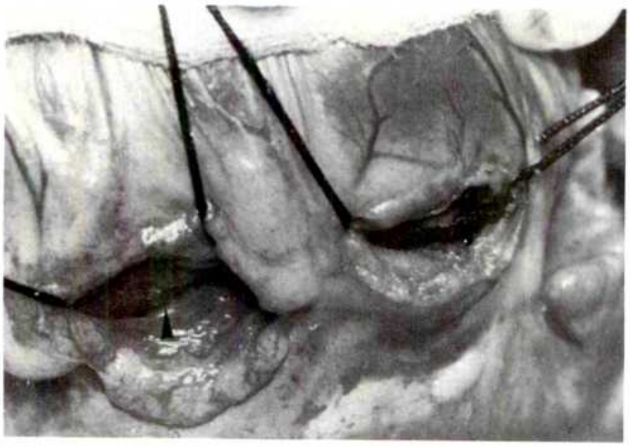

Fig 2. Close-up of direct epicardial dissection of the left atrioventricular sulcus. The left atrioventricular junction (arrow) *is well seen.*

coronary artery branches were isolated along their transverse segment to allow their mobilization during cryoablation. Coronary arteries are dissected and mobilized en bloc with their adventitial and surrounding connective tissue. Coronary artery spasm was never observed, nor was evidence of segmental ischemia in homologous vascular territory. (5) The great cardiac vein was dissected in the anterior left ventricular segment if it crossed the AV sulcus and was mobilized together with the circumflex coronary artery in some instances. (6) The AV junction was exposed. The width of dissection was determined by the location of the pathway on the epicardial map. Dissection for mid-left ventricular pathways comprised the mid-left ventricular region and adjacent left coronary fossa and left paraseptal region. Dissection for left paraseptal pathways encompassed the left paraseptal region and adjacent mid-ventricular region and posterior septal region. Using the direct epicardial approach, the left ventricular dissection was easily extended onto the posterior septal region. The right atrial–left ventricular sulcus was dissected and the posterior superior process of the left ventricle exposed without dividing the middle cardiac vein. Dissection for left anterior septal pathways included dissection of the left coronary fossa and adjacent anterior left ventricular wall. The left coronary fossa was approached by commencing the dissection on the atrial side of the sulcus and primarily identifying the left main stem and its bifurcation. Direct epicardial approach was used for the dissection of the anterior left ventricular segment.

Accessory pathway conduction was present after dissection in 1 patient with a left anterior septal pathway. Cardiopulmonary bypass was used to obtain transmural cryoablation. The heart was fibrillated and the left atrial appendage opened. The left AV junction in the left coronary fossa and the left fibrous trigone were ablated by using a cryoprobe (5 mm, −60°C, 2 minutes) through the left atrial appendage. Adjunctive cryoablation of the adjacent anterior left AV junction was carried out using epicardial application of the same cryoprobe on an empty left atrium. Epicardial cryoablation was carried out in the remaining patients using a 5-cm cryoprobe (−60°C, 2 minutes). The cryoprobe was applied on the atrial side of the AV junction while coronary arteries were kept at a distance.

One patient with associated anterior septal pathway had his left free wall pathway ablated using sling exposure without cardiopulmonary bypass; subsequently his parahissian anterior septal pathway was ablated using atrial septal endocardial dissection [10] under normothermic cardiopulmonary bypass.

The patient with associated coronary artery disease had epicardial mapping and accessory pathway ablation using sling exposure performed under normothermic cardiopulmonary bypass.

Before closing, a pair of temporary pacing wires were attached onto each cardiac chamber for postoperative electrophysiological testing.

Results

Cardiopulmonary bypass was used in 1 patient with a subendocardial left anterior septal pathway that required cardiopulmonary bypass for transmural cryoablation and in 1 other patient with coronary artery disease.

Surgical morbidity was minimal. Postoperative bleeding during the first 2 hours postoperatively was 84 ± 41.0 mL. All patients had an uneventful recovery.

Delta wave recurred intermittently in 1 patient in the early postoperative period. The accessory pathway conduction was not present during reoperation on the same day. Transmural epicardial cryoablation was carried out along the entire left AV junction using 5-cm cryoprobe (−60°C, 2 minutes) on an emptied left atrium under normothermic cardiopulmonary bypass.

There were no long-term recurrences during a mean follow-up of 7 months. All patients are free of arrhythmias and of antiarrhythmia medication.

Comment

Feasibility of the Sling Exposure

Dislocation of the heart using sling exposure was not associated with hemodynamic impairment. Sling exposure does not alter systolic ventricular function; dislocation of the heart, per se, does not occlude the vena cava or the pulmonary vein, nor does it impair tricuspid or mitral valve function. Diastolic relaxation is not altered if the sling is kept loose allowing adequate diastolic relaxation and ventricular filling. Sling exposure is quite different from exposure by an assistant's hand, which invariably squeezes the heart, prevents diastolic filling, and produces low cardiac output and hypotension. Intraoperative infusion of crystalloid solution increases cardiac filling pressure, facilitates diastolic relaxation, and prevents hemodynamic disturbances. It was routinely used.

Use of Cardiopulmonary Bypass

Cardiopulmonary bypass is associated with definite morbidity such as bleeding, generalized edema, cardiac and pulmonary dysfunction, cerebral dysfunction, and delayed febrile state [11, 12]. Lately we have documented that the serum level of creatine kinase-MB is significantly higher postoperatively in patients after ablation of accessory pathway using the epicardial approach when cardiopulmonary bypass is used (unpublished data). This result suggests that the use of cardiopulmonary bypass is associated with myocardial damage. We have observed that cardiopulmonary bypass does not permit better exposure of the left AV sulcus when the direct epicardial approach is used, which requires lesser dislocation of the ventricles. In addition, the direct epicardial approach is associated with fewer complications because the thin-walled left atrium is not extensively dissected and the coronary sinus is not mobilized [6]. No intraoperative complications were observed in our series.

Heparinization is associated with a certain degree of oozing, which does not allow precise dissection as on a dry field. Cardiopulmonary bypass is usually associated with increased postoperative bleeding [13]. We compared 2-hour postoperative bleeding in patients without cardiopulmonary bypass (sling exposure) and 35 patients with identical dissection under cardiopulmonary bypass. Patients with cardiopulmonary bypass had significantly greater 2-hour postoperative bleeding: 117 ± 83 mL versus 84 ± 41 mL ($p = 0.05$). The 24-hour drainage was less but not significantly decreased (565 ± 250 mL versus 481 ± 195 mL). These findings are consistent with the observation that, after 2 hours, the drainage is composed of serous fluid rather than blood. Cardiopulmonary bypass is kept in the operating room with the perfusionist standing by and can be rapidly attached to the patient if necessary, as already done for ablation of posterior septal pathways and right free wall pathways [6]. In our overall experience with the epicardial approach resternotomy for bleeding was performed only in patients whose operation was carried out under cardiopulmonary bypass.

Efficacy

The efficacy of epicardial cryoablation is decreased when the cryoprobe is applied on the beating heart with normal cardiac output. The circulating warm pool of blood decreases the depth of cryonecrosis [3, 14]. We have established that accessory pathway conduction should be interrupted by dissection before using epicardial cryoablation. When accessory pathway conduction is still present after dissection (subendocardial pathways) cardiopulmonary bypass was used to attain uniform transmural cryoablation. However, we have observed one early relapse of an "epicardial" pathway although such early recurrence was never observed when epicardial cryoablation under cardiopulmonary bypass, which nullifies left atrial blood flow, was used. It has been suggested that lower temperatures (−140°C instead of −60°C) should be routinely used [15].

Watanabe and associates [15] have independently developed a similar epicardial approach. Recently, they reported [15] 14 patients with left free wall pathways in whom epicardial ablation was attempted without support of cardiopulmonary bypass. The left free wall accessory pathways were exposed through a left thoracotomy in 3 patients and a median sternotomy in 11. The surgical technique combined dissection using an ultrasonic surgical aspirator and deep cryoablation (−140°C). Ten pathways were ablated without the need for cardiopulmonary bypass, whereas four pathways required the use of endocardial dissection for definite ablation.

Addendum

Since completion of the manuscript, 34 patients were operated on using the same technique with no failures and no complications.

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Management of Aortobronchial Fistula With Graft Replacement and Omentopexy

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Massive hemoptysis due to a recurrent aortobronchial fistula after repair of a thoracic aortic aneurysm developed in a 64-year-old woman. The infected aortic tissue was resected and replaced with an in situ Dacron graft covered by omentum. The patient is alive and well 15 months later.

(Ann Thorac Surg 1990;50:972–4)

Aortobronchial fistula is a rare but catastrophic complication after repair of thoracic aortic aneurysm [1–3]. Surgical management involves control of the airway, left thoracotomy, shunt or bypass, aortic suture repair or graft replacement, and lung resection. Despite excellent initial survival [4], recurrent infection, inflammation, and pulsatile erosion of the aorta into the lung may lead to recurrent aortobronchial fistula [2]. We report a case of recurrent aortobronchial fistula after thoracic aortic aneurysm repair that was successfully managed with omentopexy.

A 64-year-old hypertensive woman had a routine chest radiograph which demonstrated a descending thoracic aortic aneurysm. Arteriography showed a large, saccular aneurysm, 5 cm in diameter, protruding from the left lateral wall of the thoracic aorta (Fig 1). She underwent resection of the thoracic aneurysm with a Dacron patch repair of the lateral aortic wall. Lung tissue densely adherent to aneurysm wall required wedge resection. Microscopically, there was chronic inflammation and medial degeneration of the aorta and chronic inflammation and fibrosis of the adjacent lung. Cultures were negative. The postoperative course was uneventful and she was discharged on the seventh day.

One and one-half years later, hemoptysis requiring hospital admission developed. Chest roentgenogram, computed tomographic scan, aortogram, and triple endoscopy were normal. No further bleeding occurred and she was discharged.

One month later the patient experienced massive hemoptysis. Bronchoscopy showed active bleeding from the left lower lobe. A left thoracotomy was performed. Dense adhesions were seen between the lung and the aortic patch, and the aortopulmonary fistula was identified. The aorta was repaired with multiple pledgeted Prolene sutures. The previous patch was reinforced with another Dacron patch. All cultures were negative. The patient did well and was discharged on the seventh postoperative day.

Fourteen months later, massive hemoptysis recurred. Chest roentgenogram and computed tomographic scan showed consolidation of the left lower lobe. Arteriogram demonstrated irregularity along the previous graft. There was no extravasation. A third left thoracotomy was performed. Again, dense adhesions were encountered. Left atrial to femoral artery bypass with a Bio-Medicus pump accompanied aortic cross-clamping. The Dacron patch suture line was found to have eroded into the left lower lobe. Inflamed aorta and old patch material were resected

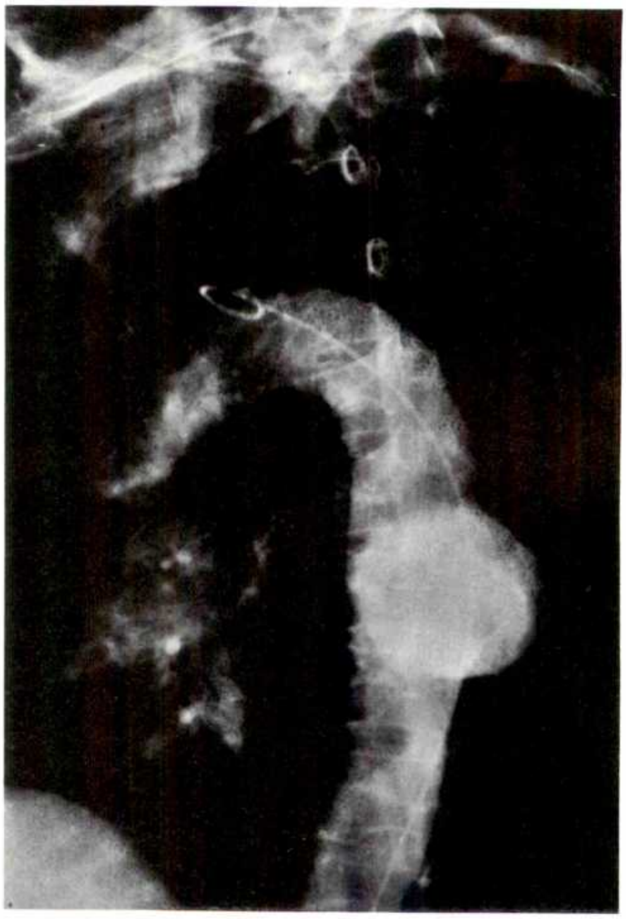

Fig 1. Preoperative arteriogram demonstrating large, saccular aneurysm of the descending thoracic aorta.

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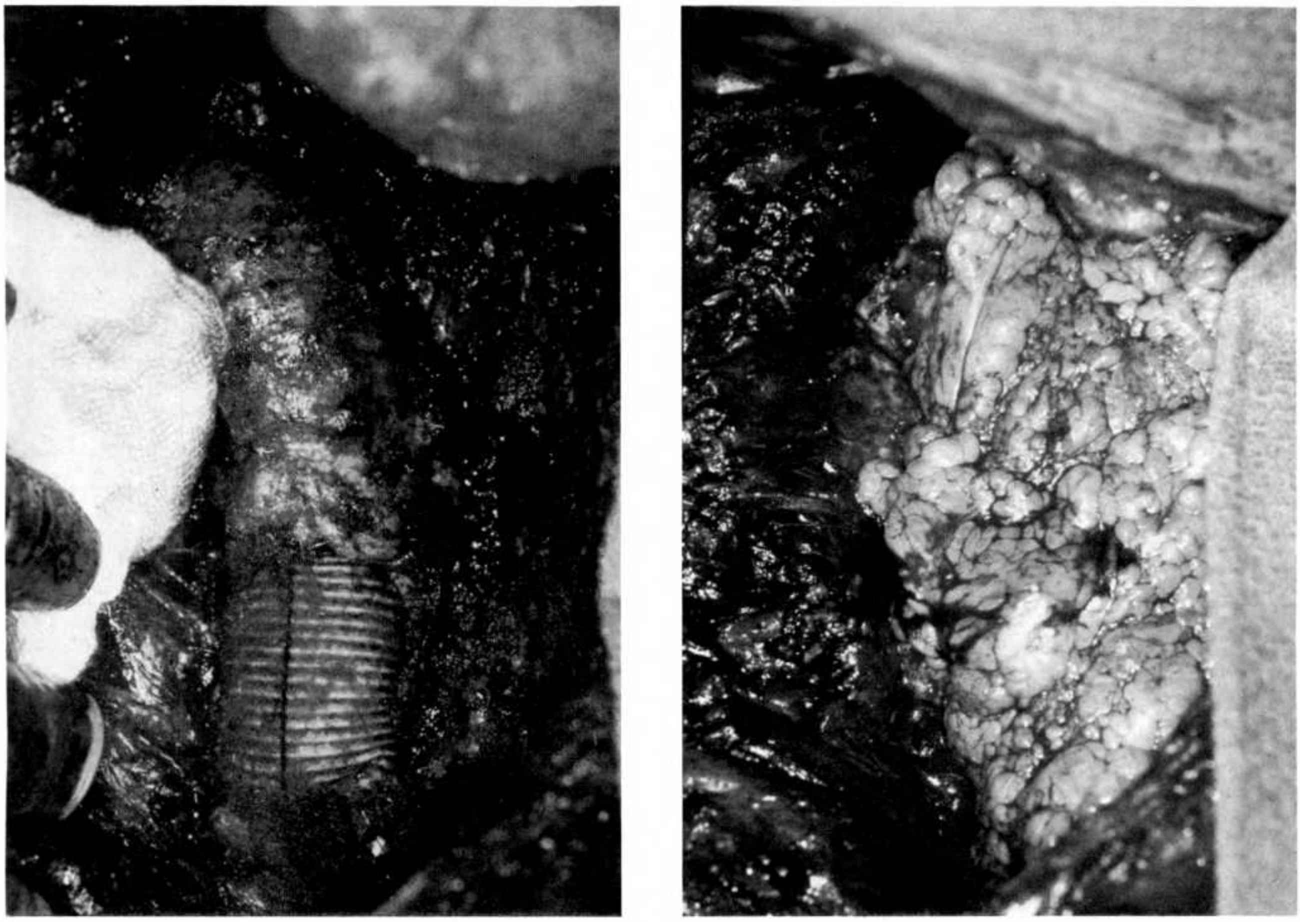

Fig 2. (A) Inflamed aorta and old patch have been resected and replaced with new graft. (B) Graft has been covered with mobilized omentum.

and replaced with an in situ 22-mm woven Dacron tube graft (Fig 2A). Adjacent lung was stapled and oversewn. The omentum was mobilized through a left subcostal incision and delivered through the diaphragm. The graft was wrapped with omentum (Fig 2B). Cultures from resected lung and aortic tissue grew *Haemophilus influenzae*. Blood cultures were negative. The patient was extubated on the second postoperative day and had an uncomplicated recovery. Intravenous antibiotics were administered for 6 weeks, the last 4 weeks on an outpatient basis. Fifteen months later, she is doing well without recurrent hemoptysis (Fig 3).

Comment

Aortobronchial fistula is a rare but often lethal complication of untreated thoracic aneurysms or repair of such aneurysms [1, 2, 4, 5]. It may occur more than a decade after successful repair of a thoracic aortic aneurysm [1]. Etiology of aortobronchial fistula includes infection and inflammation, as well as pulsatile erosion of suture line leaks into adjacent lung [3, 6]. Nearly all patients have hemoptysis, often massive and immediately life threatening, when first seen [4]. Correct premortem diagnosis occurs in less than half the cases, which is tragic as timely surgical intervention can be successful in 80% of patients undergoing operation [4]. Chest roentgenogram, computed tomographic scan, and arteriography may demonstrate an aneurysm with soilage of the lung with blood, but they do not directly show a fistula. As demonstrated by our patient, normal radiographic studies do not reliably exclude a diagnosis of aortobronchial fistula [1]. Bronchoscopy may demonstrate blood in the affected lobe but should be performed only if immediate thoracotomy is available. Preoperative preparation includes intubation with a double-lumen tube to prevent soilage of the contralateral lung.

Although surgical management of aortobronchial fistula has varied, it includes pulmonary resection and aortic repair or replacement. Limited wedge resection of adherent lung is all that is usually required, but occasionally major pulmonary resection is necessary [1, 3, 4]. The aortic repair may be by direct suture [2], resection and graft replacement [1, 2, 4, 5], or aortic resection with oversewing and extraanatomical bypass [6]. Healthy tissue, including aneurysm wall, pleura, or pericardium, had been used to separate the aortic repair from the lung [2, 5]. Symbas and associates [6] used omentum to cover

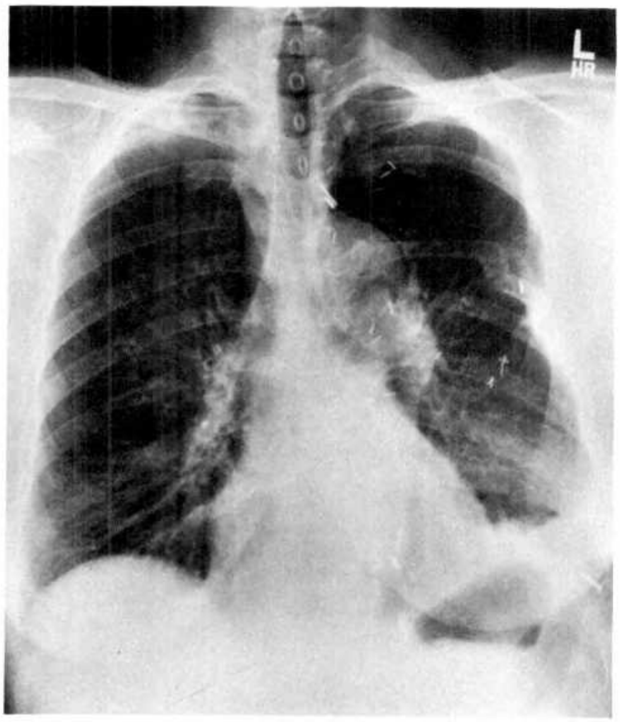

Fig 3. Chest roentgenogram 15 months after operation.

the aortic stumps after resection of an infected thoracic aorta following an orthopedic procedure.

Our patient is only the second reported case of survival after two operations for recurrent aortobronchial fistula involving an aortic graft, the first being reported by Graeber and colleagues [2]. Both patients had remarkably similar courses. The interval between aortobronchial fistulas was 12 months in Graeber's patient and 14 months for our patient. In the present case, the aortobronchial fistula recurred after direct suture repair. In retrospect, we believe that such treatment, while lifesaving, is probably inadequate in preventing recurrence. Smoldering inflammation may allow further erosion of aortic suture lines into lung. Resection of all synthetic material and adherent lung, graft replacement, and omental wrapping of the new graft appears to be a more complete alternative. The omentum has an established role in the treatment of complicated cardiothoracic problems [7]. It serves as a mechanical barrier between aorta and lung and would seem to aid in the prevention of continued or recurrent infection.

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Aortobronchial Fistula After an Aortic Operation

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A 71-year-old man with a postoperative aortobronchial fistula was successfully treated. The fistula occurred between the left lower lobe and the descending thoracic aorta, to which a distal anastomosis of a temporary bypass graft had been placed during thoracic aortic aneurysmectomy 3 years before. For saving patients with this complication, early surgical treatment during episodes of intermittent hemoptysis is important. The use of an omentum pedicle flap for the isolation of the suture line is a important adjunct.

(Ann Thorac Surg 1990;50:975–7)

Aortobronchial fistula (ABF) is a rare but highly lethal condition. If untreated, the condition is often fatal because of exsanguinating hemoptysis. Sometimes the ABF occurs as a postoperative complication after replacement of the thoracic aorta with a prosthetic vascular graft. From review of the 13 cases that have been reported, including our present case, we herein discuss the preoperative diagnosis and method of prevention and treatment of the postoperative ABF.

A 71-year-old man was admitted to our hospital on December 6, 1988, because of intermittent hemoptysis during the past 3 months. Three years before his admission, he had undergone a Dacron patch aortoplasty for an aortic aneurysm of the distal arch involving the just-proximal portion of the left subclavian artery. During aortic cross-clamping, distal perfusion was maintained by a temporary bypass graft placed between the left common carotid artery and the descending thoracic aorta. On admission, physical examination revealed a blood pressure of 140/80 mm Hg and a pulse rate of 120 beats/min. Laboratory examination showed slight anemia with a hematocrit of 0.34 and a hemoglobin level of 0.1 g/L. On chest x-ray examination, computed tomography, magnetic resonance imaging, and endoscopic ultrasonography through the esophagus, no abnormal shadows suggesting the presence of an anastomotic aneurysm were recognized. On bronchoscopic findings, ostia of the left lateral basal and posterior basal bronchus were edematous and filled with blood clots. Because the patient had a previous episode of iodine-induced shock, aortography was not performed. The characteristic feature of his intermittent hemoptysis and the findings of bronchoscopy strongly suggested the presence of an ABF at the site of the distal anastomosis of the previous temporary bypass graft, and a second operation was performed.

Selective endobronchial intubation was used in the anesthetic management. A temporary bypass graft was constructed between the right axillary artery and the left external iliac artery, and the omental flap with a vascular pedicle was prepared using a midline abdominal incision. The residual patch of the synthetic graft, which remained at the distal anastomosis of the previous temporary bypass graft in closing, had protruded into the left lower posterior lobe of the lung, and the visceral pleura was eroded because of adhesion of the pulsating mass (Fig 1). The residual graft was completely removed along with the adjacent tissue. The aortic defect was reconstructed using a Dacron patch aortoplasty. The bronchial end of the fistula was closed with suture ligature. In addition, the omental flap was spread and fixed over the aorta to isolate the suture line from the lung. The postoperative course was uneventful, and his intermittent hemoptysis has completely disappeared.

Comment

Postoperative aortobronchial fistula is documented in the literature as a rare but serious late complication of thoracic aortic operations. Only 13 cases of this disease have been reported: after thoracic aortic aneurysmectomy in 7 cases [1–4], after coarctation repair in 5 [5, 6], and after division of the persistent ductus arteriosus in 1 [7] (Table 1). Among them there was only 1 case of the ABF that occurred at the distal end of a temporary bypass graft, similar to our case [2].

Although if untreated the mortality rate for this condition is almost 100%, it is difficult to make a correct preoperative diagnosis [8]. At our institution, ABFs caused by thoracic aortic anastomotic aneurysm were encountered in 5 patients during the period from 1970 to 1988. Three patients died of massive hemorrhage before surgical treatment. One underwent an emergency operation in the hemorrhagic shock state and died from uncontrollable bleeding during operation. The only surviving patient is the one reported herein, who was operated on

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Table 1. Previously Reported Postoperative Aortobronchial Fistulas

Author	Age	Sex	Condition and Initial Operation	Onset of Episode	Main Symptom	Duration of Symptoms	Method of Diagnostic Value	Surgical Result
Davey MG (1962)	35	M	Coarctation, Ivalon graft replacement	5 y	Intermittent hemoptysis	10 w	. . .	Survived
Garrett HE (1965) [1]	62	M	Dissecting aneurysm, Dacron graft replacement	4 y	Intermittent hemoptysis	6 w	. . .	Survived
Neville WE (1966)	40	M	Traumatic aneurysm, Orlon graft replacement	10 y	Intermittent hemoptysis, substernal pain	20 w	Chest x-ray, Aortography	Survived
Boerema I (1971)	21	F	Coarctation, end-to-end anastomosis	10 m	Intermittent hemoptysis	10 w	Aortography	Survived
Kakos GS (1975)	56	F	Coarctation, repair	7 y	Intermittent hemoptysis	8 w	Bronchoscopy	Survived
Vasko JS (1977)	22	M	Traumatic aneurysm, Dacron graft repair	3 y	Massive hemoptysis	?	. . .	Survived
Graeber GM (1980) [2]	61	M	Thoracic aneurysm, graft replacement with temporary bypass graft between left atrium and descending aorta	10 y	Intermittent hemoptysis (episode 1)	6 w	Chest x-ray	Survived
				11 y	Intermittent hemoptysis (episode 2)	3 w	Chest x-ray	Survived
Singh AK (1982) [5]	24	F	Coarctation, Dacron patch repair	4 y	Intermittent hemoptysis	?	Aortography, bronchoscopy	Survived
Uehara K (1983) [6]	26	M	Coarctation, Teflon graft replacement	13 y	Intermittent hemoptysis	4 d	Aortography, chest x-ray	Survived
Oldham KT (1983) [3]	36	M	Traumatic aneurysm, Teflon graft replacement	15 y	Massive hemoptysis	. . .	Bronchoscopy	Died
Wood AE (1984) [7]	11	F	PDA, division of the ductus	9 y	Massive hemoptysis	. . .	Chest x-ray, CT, bronchoscopy	Survived
Coblentz CL (1988) [4]	43	M	Thoracic aneurysm, synthetic graft replacement	10 y	Intermittent hemoptysis	?	Aortography, CT, bronchoscopy	Survived
Coblentz CL (1988) [4]	71	M	Thoracic aneurysm, synthetic graft replacement	3 y	Intermittent hemoptysis	?	Bronchoscopy	Died
Ishizaki Y (present case)	71	M	Thoracic aneurysm, Dacron graft patch plasty with temporary bypass between left carotid artery and descending thoracic aorta	3 y	Intermittent hemoptysis	12 w	Bronchoscopy	Survived

CT = computed tomography; PDA = patent ductus arteriosus.

between episodes of intermittent hemoptysis. All patients except 2 who were operated on immediately after massive hemoptysis had a period of intermittent hemoptysis that ranged from 4 days to 20 weeks (mean, 10.3 weeks), and the operations were successfully performed during this period in 9 patients (see Table 1).

On the radiological examinations including aortography, fistulous communications were not confirmed in all cases because they were covered with blood clots [2, 7]. Only bronchoscopy had some value in detection of the site of entrance of the fistula into the bronchial tree [2–5, 7], but this examination should be undertaken with extreme care because it presents a danger of accidentally dislodging the clot acting as a stopcock of the fistula

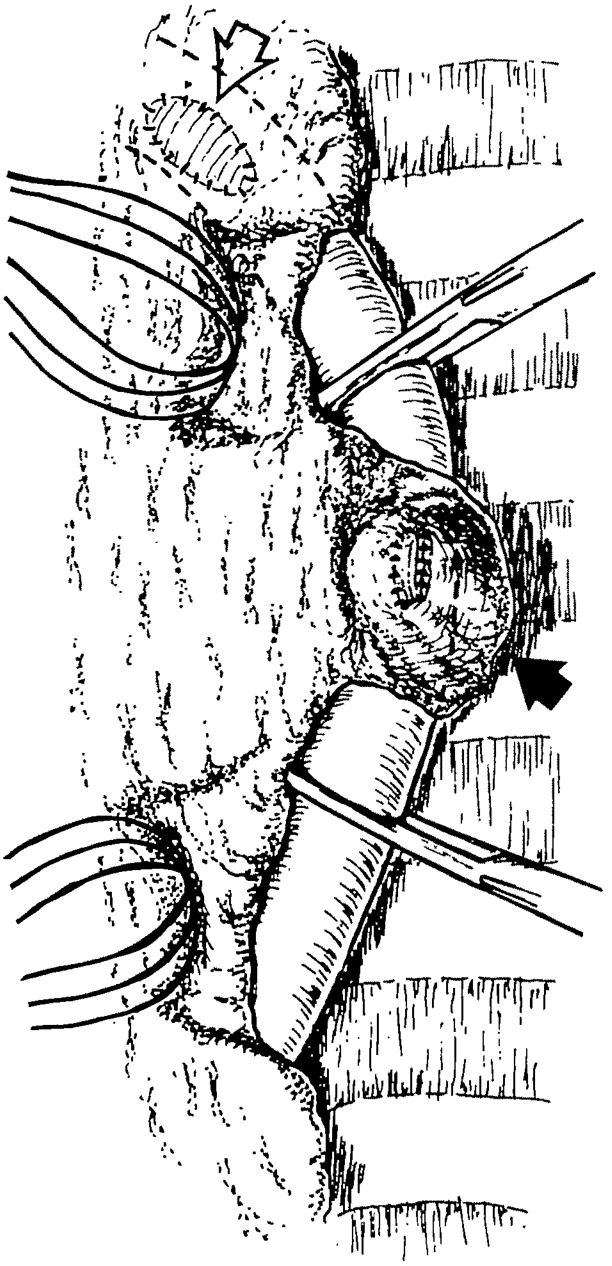

Fig 1. Operative findings. A Dacron patch aortoplasty that was undergone during the previous aneurysmectomy is shown (white arrow). *Residual patch of the temporary bypass graft protruded into the left lower posterior lobe of the lung* (black arrow). *The fistulous mass was left untouched until proximal and distal aortic cross-clamping was finished, then residual graft was completely removed along with the adjacent tissue.*

which, in turn, results in massive bleeding during the examination. We thus emphasize the importance of early operation during the asymptomatic interval even if confirmation of the diagnosis of an ABF cannot be obtained by radiologic examination.

Operative treatment of the fistulous mass is very problematical and requires meticulous consideration. In anesthetic management, selective endobronchial intubation is recommended to control possible intraoperative hemorrhage [1, 2, 7]. Management of the aortic end of the fistula can be accomplished by patch closure [8], direct suture [7], grafting [4, 6], and subclavian artery flap repair [5]. The bronchial end of the fistula is closed with direct suture, or with partial or total pneumonectomy. As a preventive procedure to the recurrence of the fistula, protection and isolation of the suture line with living tissue, such as the pleural flap [2, 6], adventitial flap [2], intercostal muscle flap [1], or omental flap (as used in our patient), is considered to be beneficial. Since our experience with this case of aneurysmectomy of the thoracic aorta, a temporary bypass graft has now routinely been made between the right axillary artery and the left external iliac artery to avoid direct contact between the closing anastomosis and the lung.

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Congenital Tracheoesophageal Fistula Associated With Carcinoma of the Lung in an Adult

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A rare case of adult congenital H-type tracheoesophageal fistula was diagnosed. Subsequently, at operation, large cell, undifferentiated carcinoma of the right middle lobe with extension to the right lobe and adherence to the diaphragm was documented. The diagnosis, surgical intervention, and 4-year follow-up are presented.

(Ann Thorac Surg 1990;50:978–9)

A 40-year-old man who had a history of multiple recurrent episodes of pneumonia that started shortly after birth was seen with symptoms of pneumonitis. He appeared to be well developed, well nourished, in fairly good health, and in no acute distress. Physical examination and past history were unremarkable. He had smoked about one pack of cigarettes daily but had stopped some 9 years previous. Family history revealed carcinoma (nonspecified) on the side of his maternal grandmother.

Chest roentgenograms showed right middle lobe infiltrate or atelectasis, old granulomatous disease, and a heart size within normal limits. Transnasal fiberoptic bronchoscopy had been performed under local anesthesia to rule out pneumonitis or neoplasm. The bronchoscope was passed through the vocal cords into the trachea where a well-defined hole, approximately 6 mm in diameter, was seen in the posterior tracheal wall. The bronchoscope was advanced through the right main bronchus. The right middle lobe showed moderate hyperemia and tenacious mucoid secretions with inflammatory narrowing of the medial and lateral segments. The right lower lobe showed substantial narrowing at the superior segment with severe hyperemia, edematous changes, and mucoid secretions. Examination of the basilar segment of the lower lobe, however, was unremarkable. The bronchoscope was withdrawn into the trachea where it was passed through the 6-mm orifice in the posterior tracheal wall and into the esophagus. The esophagus was traversed to midsection before the bronchoscope was withdrawn.

Operation was performed through a posterolateral thoracotomy incision made at the level of the T5-6 interspace. In the chest, minimal adhesions were noted at the upper lobe but the middle and lower lobes and a section of the diaphragm showed marked scarring and adhesions. A large mass was found in the middle lobe.

Attention was then turned to the tracheoesophageal fistula. The tracheoesophageal fistula was found easily after opening the mediastinal pleura. There was no evidence of scarring in the area of the fistula and dissection was fairly easy. Care was taken to preserve the blood supply to both the trachea and esophagus, and the fistula was cut in such a fashion as to leave enough esophageal wall to close the defect in the trachea. The esophagus was closed primarily with no tension. A flap of pleura was developed and interposed between the trachea and esophagus at the level of the fistula.

Dissection of the middle lobe and biopsy with frozen section of the mass disclosed large cell, undifferentiated carcinoma. Upon excision, the entire mass showed the same, and it was believed the lower lobe should also be removed owing to the adherence of the mass. The area on the diaphragm where the tumor was adherent was also excised with wide margins and closed primarily under no tension. Pathological study showed this area to be inflammatory with no evidence of tumor.

The patient did well after operation. Six days postoperatively a barium swallow with fluoroscopy was performed. There was no evidence of extravasation and no fistulous tract was apparent. The esophagus was normal.

Almost 4 years after operation the patient continues to work and live well without episodes of pneumonia and no evidence of recurrent cancer.

Comment

An adult congenital H-type tracheoesophageal fistula is rarely seen, with only about 20 case studies having been reported since 1929 [1, 2]. The H-type of tracheoesophageal fistula, which is not associated with esophageal atresia, is a rare anomaly that occurs approximately once in 100,000 births [3]. Of 14 cases reviewed in *Thorax* in 1982 [3], only 1 was noted to have associated carcinoma of the lung. This was an incidental finding on autopsy.

Adult patients usually are first seen with chronic recurrent pneumonia, hemoptysis, or symptoms of chronic pulmonary infection. The work-up is usually indicated because of these symptoms or because of roentgenographic findings indicative of infection or abscess in the lung. In the case presented, a bronchoscopy diagnosed the fistula and therefore a barium swallow was not needed. The approach taken was a right posterolateral

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thoracotomy as the fistula was clearly in the chest and there was also disease in the lung, the exact nature of which had to be determined. There have been, of course, other approaches to the repair of these fistulas, and these include a cervical approach through a lower transverse collar incision with or without partial upper median sternotomy [2].

In this case, identification of the fistula at operation was relatively easy and accomplished with minimal dissection. Nevertheless, the literature reports difficulty in identifying the fistula at operation, and various methods have been described to accomplish this including passing a guidewire through the fistula preoperatively [4] via the bronchoscope as well as opening the trachea for inspection [2].

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Notice From the American Board of Thoracic Surgery

The American Board of Thoracic Surgery began its recertification process in 1984. Diplomates interested in participating in this examination should maintain a documented list of the operations they performed during the year prior to application for recertification. This practice review should consist of 1 year's consecutive major operative experiences. (If more than 100 cases occur in 1 year, only 100 need to be listed.) They should also keep a record of their attendance at approved postgraduate medical education activities for the 2 years prior to application. A minimum of 100 hours of approved CME activity is required.

In place of a cognitive examination, candidates for recertification will be required to complete both the general thoracic and cardiac portions of the SESATS IV syllabus (Self-Education/Self-Assessment in Thoracic Surgery). It is not necessary for candidates to purchase SESATS IV booklets prior to applying for recertification. SESATS IV booklets will be forwarded to candidates after their applications have been accepted.

Diplomates whose 10-year certificates will expire in 1993 may begin the recertification process in 1991. This new certificate will be dated 10 years from the time of expiration of the original certificate. Recertification is also open to any diplomate with an unlimited certificate and will in no way affect the validity of the original certificate.

The deadline for submission of applications is July 1, 1991. A recertification brochure outlining the rules and requirements for recertification in thoracic surgery is available upon request from the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Bronchial Anomaly of the Right Upper Lobe

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This case report presents a rare anomaly of right upper lobe bronchial anatomy. During routine right upper lobe resection for carcinoma, a common right upper and middle lobe bronchus was found. The resection was completed as a right upper and middle bilobectomy. Knowledge of this uncommon variant was beneficial in performing the pulmonary resection. A review of the literature is presented.

(Ann Thorac Surg 1990;50:980–1)

Previous reports have described various anatomical anomalies of the bronchovascular structure of the human lung [1–4]. These have been estimated to occur in approximately 3% of the general population [2]. A general appreciation of these anomalies is valuable to the diagnostician as well as to the thoracic surgeon.

Aberrant bronchial anatomy of the right upper lobe most commonly is the result of supernumerary or anomalous segmental bronchi originating from the lateral wall of the trachea, at or near the level of the carina [3]. Less frequently observed anomalies can be the result of accessory bronchi originating from the bronchus intermedius [5]. Finally, one of the rarest anomalies of the right upper lobe is a common bronchus giving rise to both the right upper and middle lobes. The present case report describes a patient with this rare anatomical variant.

A 68-year-old patient had a solitary pulmonary nodule discovered on routine chest roentgenogram. Bronchoscopy revealed no endobronchial lesions and what was thought to be a low take-off of the right upper lobe bronchus with multiple segmental bronchi. A middle lobe bronchus was not clearly identified. Transbronchial biopsy was positive for an adenosquamous carcinoma.

At operation, a 3-cm tumor in the anterior segment of the right upper lobe encroaching on the minor fissure was found. Dissection revealed that the right upper and middle lobe bronchi originated from a common bronchus. Due to tumor invasion of the middle lobe, this common bronchus was divided, completing both a right upper and middle lobectomy.

Postoperative dissection of the resected specimen demonstrated a right upper lobe consisting of three normal anatomical divisions with the correct orientation as well as a right middle lobe, which had divisions resembling the inferior and superior segments of a lingula. The common lobar bronchus measured 2 cm in length and originated from the right main bronchus (Fig 1).

Comment

Since the commencement of thoracic surgery, there has been clinical interest in identifying the anatomy of the tracheobronchial tree. Bronchography, detailed autopsy analysis, and meticulous operative dissections have aided in defining both common anatomy as well as its relatively infrequent variations.

Bronchial anomalies occur in approximately 2% to 3% of the general adult population, predominantly involving the right upper lobe. The most frequently observed anomalies are supernumerary, displaced segmental or lobar bronchi originating from the lateral trachea or right main bronchus. These aberrant segmental bronchi are typically from the apical segment of the right upper lobe, though other segments can be involved.

In sheep, pigs, and cows, among other animals, a tracheal bronchus normally develops proximal to the carina and supplies the apical segment of the right upper lobe [6]. However, in humans this occurs in only approximately 5% of embryos and is observed in only 2% of the general adult population [7].

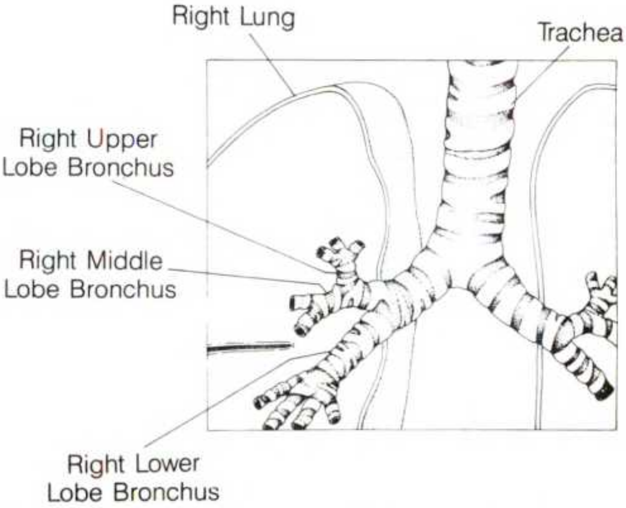
Right Lung
Trachea
Right Upper Lobe Bronchus
Right Middle Lobe Bronchus
Right Lower Lobe Bronchus

Fig 1. Bronchial tree with anomalous right upper lobe bronchial anatomy. Note the common right upper and middle lobe bronchus. This anatomy, along with the position of the tumor, dictated a right upper and middle lobectomy for a curative resection.

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Clinically, the anomalies of the tracheobronchial tree are usually asymptomatic and are discovered as incidental findings at bronchoscopy, bronchography, or surgical resection. However, occasionally these patients may have a persistent cough, stridor, resistant right upper lobe pneumonia, or bronchiectasis that is only in retrospect attributed to their aberrant bronchial anatomy. Although occasionally associated with pulmonary infections, these anomalies are not thought to predispose to malignant transformation.

Depending on the clinical presentation, the treatment for these anomalies ranges from observation to surgical resection. Those patients who have clinically silent bronchial anatomical variants need only be observed. Those with persistent or progressive pulmonary compromise secondary to complications from this aberrant anatomy benefit from a pulmonary resection.

The present case report describes one of the more rare anomalies of the right upper lobe consisting of a common right upper and middle lobe bronchus resembling the mirror image of the normal left upper lobe. Although this anomaly was clinically undetected before operation, an appreciation of this anatomical variant involving the tracheobronchial tree is important for accurate preoperative bronchoscopic evaluation as well as subsequent safe pulmonary operation.

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Notice From the American Board of Thoracic Surgery

The part 1 (written) examination will be held at the Hyatt-Regency, Dallas Fort Worth Airport, Dallas TX, on February 16, 1992. The closing date for registration is August 1, 1991.

To be admissible for the part II (oral) examination, a candidate must have successfully completed the part I (written) examination.

A candidate applying for admission to the certifying examination must fulfill all the requirements of the board in force at the time the application is received.

Please address all communications to the American Board of Thoracic Surgery, One Rotary Center, Suite 803, Evanston, IL 60201.

Management of Mycotic Rupture of the Ascending Aorta After Heart-Lung Transplantation

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Horst von der Hardt, MD, and Frauke Manthey-Stiers, MD

Division of Thoracic and Cardiovascular Surgery, Department of Pediatric Pulmonology, and Department of Medical Microbiology, Hannover Medical School, Hannover, Federal Republic of Germany

A 19-year-old woman underwent heart-lung transplantation for obstructive bronchiolitis. Eleven days later mycotic rupture of the ascending aorta occurred. Emergency cardiopulmonary bypass was instituted via the femoral vessels and the chest was reopened under cardiac massage. Perforation of the aorta at the site of insertion of the cardioplegic cannula in the donor aorta was seen. Under circulatory arrest the infected area was excised, a pericardial patch plasty was performed, and the region was covered with a muscle flap. Postoperatively, *Candida albicans* was found in the excised specimen of the aorta, in drainage fluid, and in the bronchoalveolar lavage. High doses of antimycotics were administered intravenously and for irrigation of the mediastinum, which resulted in an eradication of fungi in all cultures 7 days later. Six weeks after reoperation the patient was discharged home, and she remains well 9 months postoperatively.

(Ann Thorac Surg 1990;50:982–3)

In recent years heart-lung transplantation has developed from an experimental model to a widely accepted treatment for terminal cardiopulmonary disease with increasing survival rates at an acceptable individual risk [1]. Complications such as infection and rejection, however, are frequently observed postoperatively, requiring an enormous effort in diagnosis and therapy [2]. We present a case of mycotic rupture of the donor aorta 11 days after heart-lung transplantation.

Terminal cardiopulmonary failure from obliterative bronchiolitis developed in a 19-year-old woman. Several episodes of right heart failure had to be treated and constant oxygen supply was necessary due to severe hypoxemia at rest. She therefore qualified for heart-lung transplantation. On July 17, 1989, suitable donor organs were available and heart-lung transplantation was routinely performed.

Preoperatively, bronchoalveolar lavage of the donor lungs showed gram-negative bacteria. Postoperatively, *Candida* had grown in the culture of the donor bronchoalveolar lavage as well as in the solution used for the storage of the graft during transportation. Due to the absence of pseudomyceles, activation of fungi was considered to be low and antifungal treatment therefore was not given.

The initial postoperative period was uneventful. The patient was extubated on the second postoperative day. Mobilization, nutrition, and triple-drug immunosuppression were initiated routinely. On day 11, hypovolemic shock developed. The chest roentgenogram showed a wide mediastinum and bilateral pleural effusions compared with the film taken the day before. Echocardiography performed at the same time revealed fluid in the posterior mediastinum and both pleural cavities.

A decrease in the blood hemoglobin level from 94 to 76 g/L (9.4 to 7.6 g/dL) was measured and the patient was transferred to the operating room immediately. After induction of anesthesia, profound shock occurred with no arterial pressure measurable in the radial artery. Emergency cannulation of the femoral vein and artery was performed, cardiopulmonary bypass was instituted, and 2,500 mL of asanguineous prime solution of the pump was infused while circulation was maintained by external cardiac massage. After reopening of the sternotomy wound, bleeding in the area of the ascending aorta was seen.

Under digital compression and additional volume replacement circulation could be stabilized on cardiopulmonary bypass. Rupture of the aortic anastomosis was suspected and we decided to perform the repair under deep hypothermia and circulatory arrest to avoid cardioplegic arrest of the transplanted heart. Because infection seemed to be the most probable cause for the aortic rupture 11 days after heart-lung transplantation, the abdomen was opened during cooling and the omentum was prepared for subsequent coverage of the infected area. Masses of clotted blood were removed from both pleural cavities. At 20°C (rectal temperature) cardiopulmonary bypass was stopped and the ascending aorta was inspected. Surprisingly, the rupture had occurred at the previous cardioplegic cannula insertion site in the donor part of the vessel, while the anastomosis was intact. The perforated region was then excised and replaced with a 2 × 2-cm pericardial patch. The suture line was secured with fibrin glue mixed

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with antibiotics (bacitracin, neomycin). At this time, it was realized that the omentum pedicle was no longer viable because of thrombosed arterial and venous vessels. As an alternative, the left rectus abdominis muscle was prepared, flapped into the mediastinum, and fixed to the ascending aorta with both sutures and fibrin sealant. After application of mediastinal irrigation drainage and pleural tubes the patient was weaned from cardiopulmonary bypass without problems. Chest and abdomen were closed in a routine fashion, and the patient was transferred to the intensive care unit. She was extubated 8 hours later.

The next day, culture results revealed infection of the excised area of the ascending aorta by *Candida albicans*. An antimycotic drug regimen was initiated using amphotericin B (increasing dosage from 0.3 to 1 mg $\cdot$ kg^{-1} $\cdot$ day^{-1}, intravenous) and flucytosine (1.5 g every 6 hours, intravenous) for 6 weeks. Additionally, the mediastinum was irrigated with amphotericin B (30 mg/day). The antibacterial treatment was performed initially with ceftazidime, azlocillin, and tobramycin, and later with ceftazidime alone. From postoperative day 7 on culture for *Candida* became repeatedly negative in the drainage fluid. Therefore irrigation was discontinued and the chest tubes were removed 2 days later. *Candida* antigen titer rose from 1:2 to 1:8 14 days after the event and eventually decreased to 1:2. *Candida* antibody titer reached a maximum of 1:10,240 14 days after the reintervention and continuously decreased thereafter.

The further postoperative course was complicated by repeated episodes of cardiopulmonary rejection and cytomegalovirus disease requiring prolonged treatment with ganciclovir and hyperimmunoglobulins.

From postoperative day 51 on the patient was constantly improving, and she was discharged on postoperative day 67. She has been in good condition since then with good organ function and no remaining signs of infection 9 months after heart-lung transplantation.

Both computed tomography and nuclear magnetic resonance imaging of the chest showed a normal size and shape of the ascending aorta with a viable muscle flap behind the sternum.

Comment

Among numerous infectious complications occurring in recipients of organ transplantation, mycotic blood vessel involvement is rare [3]. Cases reported so far have been associated with a high mortality [4, 5]. In heart-lung transplantation rupture of the aortic anastomosis has been observed and was usually a sequela of an infected tracheal suture line (personal communication with three different transplantation centers). This was not the case in our patient, and we attribute this to the use of the pericardial flap plasty [6]. Others have used the omental pedicle wrap to secure healing of the airway anastomoses in lung and heart-lung transplantation [7]. This procedure, however, also would not have prevented the complication observed, because the site of the aorta involved would not have been covered.

In our opinion the keystones in the successful management of the case reported are early recognition of the perforation and immediate surgical intervention, coverage of the infected area with viable tissue [7], and immediate microbiological diagnosis with close follow-up monitoring and adjustment of antimycotic treatment [8]. We highly recommend the use of viable, well-vascularized tissue such as muscle flap or omentum to provide the infected area with sufficient blood supply to enhance microbiological resistance [6, 8]. It remains unclear, but topical use of antimicrobial agents at the time of operation as well as via irrigation thereafter may also have contributed to prevent reinfection of the mediastinum.

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Squamous Carcinoma Metastatic to the Sternum

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Divisions of Cardiothoracic and Plastic Surgery, Department of Surgery, St. Louis University Medical Center, St. Louis, Missouri

A 63-year-old man had a 10 × 16-cm sternal mass 18 months after a second aortocoronary bypass operation. The resected lesion was a metastatic tumor of squamous histology. No primary tumor was found. The diagnostic work-up and treatment options are presented.

(Ann Thorac Surg 1990;50:984–6)

Carcinoma of unknown primary site is the initial diagnosis in 3% to 15% of patients with documented malignancy [1]. Squamous cell carcinomas are relatively rare in this setting, accounting for only 14% of such lesions, with adenocarcinoma and undifferentiated carcinoma occurring far more frequently [1, 2]. We present a case of metastatic squamous carcinoma seen as a sternal tumor with no identifiable primary lesion. The diagnostic work-up and treatment options are discussed.

A 63-year-old man had a 4-month history of an enlarging sternal mass. He had undergone his second aortocoronary bypass procedure for recurrent angina 18 months earlier. His postoperative course was uncomplicated and his sternal wound had healed without incident. The mass was nontender and he had no respiratory or gastrointestinal symptomatology. He had a 30-pack/year history of cigarette smoking and had been a heavy cigar smoker over the past 20 years. There was no history of fevers, chills, or sternal trauma.

On examination, a 10 × 16-cm firm, nontender mass was noted over the mid-sternum. There were no pulsations or bruits detected in the mass, and the overlying skin was normal. Examination of the heart, lungs, and abdomen revealed no abnormality and there was no lymphadenopathy.

He was admitted for further evaluation. Work-up revealed a hemoglobin level of 85 g/L (8.5 g/dL) and a white blood cell count of $15.2 \times 10^9/L$ ($15,200/\mu L$). Serum electrolyte levels, serum enzyme levels, and urinalysis were all normal. Roentgenographic and computed tomographic studies of the chest revealed sternal erosion in association with a presternal soft tissue density (Fig 1). No lung or mediastinal lesions were noted. The differential diagnosis at this time included sternal osteomyelitis, infected hematoma, pseudoaneurysm, and neoplasm. Cardiac catheterization was performed; injection of all cardiac chambers, great vessels, and vascular grafts revealed no extravasation suggestive of pseudoaneurysm.

Operative exploration demonstrated a lobular 10 × 12 × 16-cm mass that replaced the sternum. Palpation revealed areas of firm tissue abutting fluctuant regions within the mass. The sternum, costal cartilages, and medial one quarter of the pectoralis major muscles were excised en bloc (Fig 2). Because of the prior sternotomy procedures, the deep surface of the sternum had to be dissected off the epicardial surface of the right ventricle to which it was adherent. Examination of the cut gross specimen revealed a focally cystic tumor that infiltrated the sternum. Microscopic examination of a frozen section demonstrated squamous carcinoma. On review of a permanent section the tumor was noted to replace the sternal bone marrow and to erode the superficial and deep tables of the sternum (Fig 3). After excision it was noted that a portion of the epicardial surface of the right ventricle appeared granular and uneven, and a biopsy was performed. A frozen section of this biopsy specimen also revealed squamous carcinoma. The chest wall defect was bridged using Prolene (Ethicon, Somerville, NJ) mesh. The subcutaneous dead space was filled with an omental flap based on the right gastroepiploic vessels and by medial advancement of the right pectoralis major muscle based on the thoracoacromial pedicle. Subcutaneous drains were placed and the skin was closed.

The patient was discharged on the sixth day after operation following an uncomplicated postoperative

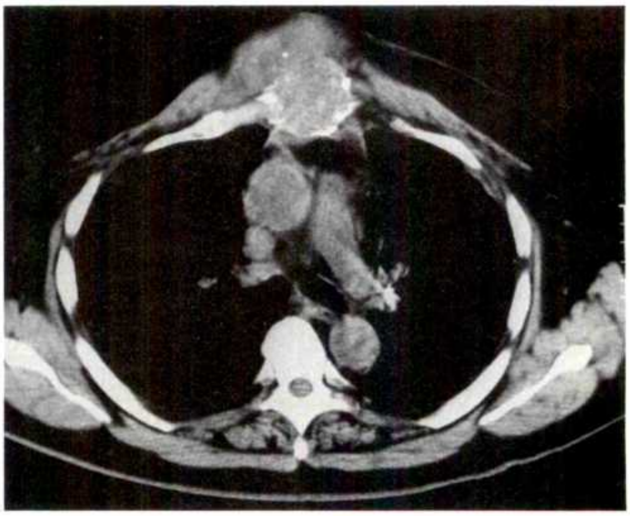

Fig 1. Computed tomogram of the chest shows presternal soft tissue density with destruction of the sternum.

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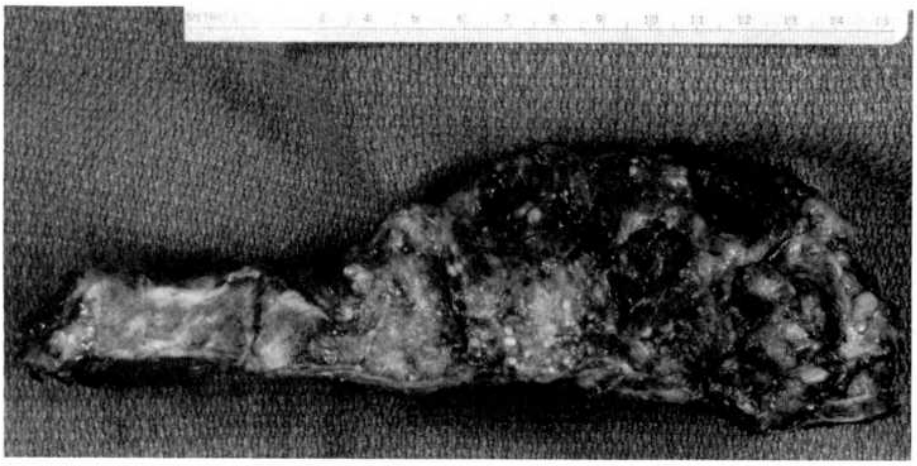

Fig 2. Sternal tumor with en bloc resection of sternum and costal cartilages.

course. After discharge, laryngoscopy, bronchoscopy, and esophagoscopy were performed and were all normal. In the absence of an identifiable primary tumor no adjuvant chemotherapy was recommended. The patient has undergone a course of high-dose irradiation to the mediastinum to treat residual carcinoma remaining on the surface of his right ventricle.

Comment

The great majority of tumors found in the sternum are malignant. The tumors found most frequently are soft tissue sarcomas, but neoplasms of reticuloendothelial origin (plasma cell tumors, myeloma) may occasionally arise at this site. The sternum can also be the site of hematogenous spread of metastases. Although this occurs infrequently, there are rare reports detailing resection of solitary but symptomatic sternal metastases for thyroid carcinoma and hypernephroma [3]. Even more infrequent is the appearance of a metastatic sternal lesion from a primary tumor of unknown origin. The tumor in this case was metastatic as cells of squamous differentiation are not present in the sternum. Pathological examination of the specimen reveals diffuse marrow replacement, suggesting the tumor may have initiated its growth there. Squamous carcinoma metastatic to bone marrow is an exceedingly rare lesion. In a recent review of more than 25,000 bone marrow biopsy specimens, only 25 specimens were metastases from unknown primary tumors, and none of these were of squamous histology [4].

A vigorous search for the primary tumor was performed. Because of the history of cigarette and cigar smoking, an oropharyngeal or pulmonary malignancy seemed likely as a primary tumor; however, careful postoperative cervical palpation, oropharyngeal examination, direct laryngoscopy, bronchoscopy, and esophagoscopy

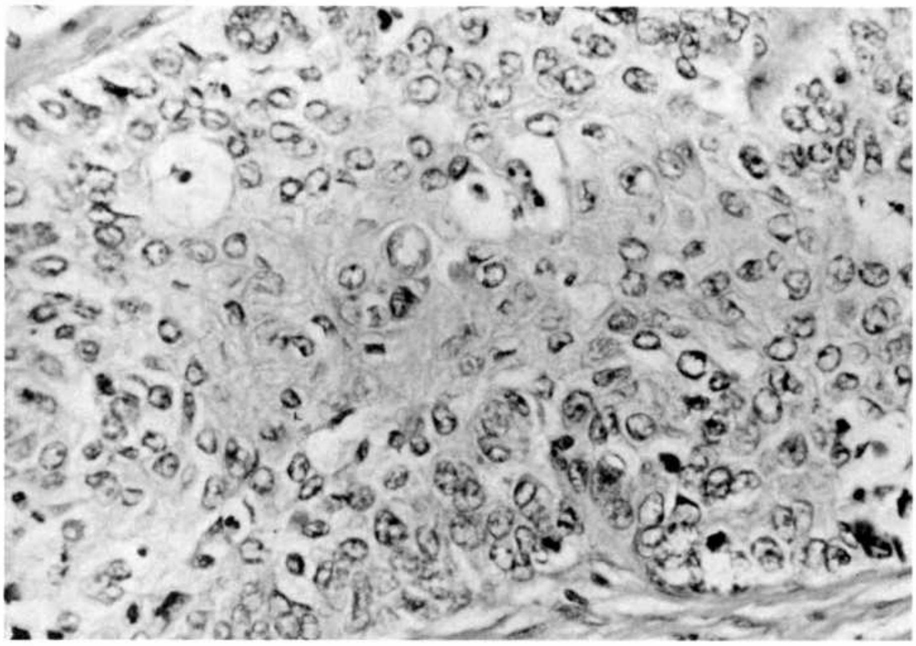

Fig 3. Histological section of the sternal tumor shows malignant squamous cell morphology. (Hematoxylin and eosin, ×400 before 30% reduction.)

failed to reveal any pathology. Retrospective inspection of the preoperative computed tomographic scan of the chest failed to reveal any pulmonary or mediastinal pathology other than the sternal tumor. It should be noted that there has been some criticism of resources and energies expended in an effort to pinpoint the primary site when presented with a metastasis of unknown origin [1, 5, 6]. In one series of 302 consecutive autopsies, 15.5% of the patients with metastatic disease never had the primary tumor site identified. However, the survival rate of these patients was no different than that of patients in whom the site of primary tumor was identified before patient death [1]. Although identification of an oropharyngeal or lung primary tumor in this patient may have allowed other treatment options, it is uncertain whether such actions would yield a substantial increase in survival.

Consideration has been given to adjuvant therapy. Although there is some indication that certain types of metastatic carcinoma from unknown primary tumors may respond to combination chemotherapy, no single or combination chemotherapy regimen has yet proved useful in extending survival in patients with squamous carcinoma from an unknown primary site [7]. Radiation therapy has been minimally successful in such clinical situations, although occasional benefit has been demonstrated. Patients with metastases to well-defined anatomical locations lend themselves to locoregional radiation therapy and can have up to 40% 3-year survival rates [5].

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REVIEW OF RECENT BOOKS

Preoperative Cardiac Assessment

Edited by Dennis T. Mangano, PhD, MD
New York, J.B. Lippincott, 1990
174 pp, illustrated, $38.00

Reviewed by Julie A. Swain, MD

This monograph from the Society of Cardiovascular Anesthesiologists is a well-written and concise description of the assessment of the patient's prior cardiac disease. The contributors are from multiple institutions and their assessments and recommendations are generally applicable.

The book is divided into five chapters. The first discusses ischemic heart disease and includes the preoperative evaluations of electrocardiography and echocardiography, with especially strong sections on nuclear scanning methods and cardiac catheterization. Routine and nonroutine diagnostic tests are described for the evaluation of the patient with ischemic heart disease. The second chapter discusses the patient with aortic and/or mitral valvar heart disease and discusses the pathophysiology of the disorders as well as the risk factors for anesthesia for both cardiac and noncardiac operations. The third chapter deals with congenital heart disease and includes a complete description of the most common congenital defects, the type of reparative operations, and the subsequent risk for anesthesia and noncardiac operative procedures.

The fourth chapter is most useful for general surgical practitioners and housestaff. It covers the preoperative assessment of the patient with heart disease undergoing noncardiac thoracic and nonthoracic surgical procedures. The discussion of spirometry and exercise studies is excellent. This chapter is useful for preliminary assessment of patients with heart disease to determine whether further specialist consultation is needed. The final chapter outlines the chronic preoperative cardiac medications that are commonly encountered. There is a short discussion of antiarrhythmic drugs, digitalis preparations, monoamine oxidase inhibitors, and other medications that are important pharmacologically.

In summary, this book presents a concise assessment of the patient with cardiac disease. Each of the chapters has a well-edited reference list. This monograph is aimed at general practitioners as a review but is especially useful for beginning housestaff in anesthesia, surgery, and medicine. It is useful as a review for those not involved in the day-to-day treatment of patients with cardiac disease.

Las Vegas, Nevada

Left Recurrent Laryngeal Nerve Paralysis: An Unusual Presentation of Histoplasmosis

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A case of histoplasmosis seen as left recurrent laryngeal nerve paralysis in a 49-year-old man is described. The patient had roentgenographic findings of a solitary, noncalcified left upper lobe mass and mediastinal adenopathy. Tissue diagnosis of histoplasmosis was obtained using a thoracotomy, allowing institution of appropriate treatment.

(Ann Thorac Surg 1990;50:987–8)

The majority of cases of histoplasmosis are asymptomatic. In endemic areas nearly 90% of the population has been exposed to *Histoplasma capsulatum*. The number of serious cases diagnosed and requiring treatment is less than 1% [1]. The majority of patients manifest only radiological signs of pulmonary infection. Symptomatic patients usually have an influenzalike illness. A hyperreactive immune response to *H capsulatum* can cause severe scarring with fibrotic encapsulation of structures in the mediastinum [2]. The signs and symptoms of pulmonary and mediastinal histoplasmosis can be difficult to distinguish from a malignancy. We report a case of histoplasmosis with an unusual symptom of hoarseness secondary to left recurrent laryngeal nerve injury. The radiological findings included a noncalcified left lung mass combined with mediastinal adenopathy.

A 49-year-old man had a 2-month history of hoarseness. Indirect laryngoscopy demonstrated unilateral vocal cord paralysis. A chest roentgenogram showed a mass in the aortopulmonary window (Fig 1). Computed tomography confirmed this mass plus a 1¼ × 1-cm noncalcified mass located in the left upper lobe of the lung (Fig 2). The patient had a 48-pack/year history of cigarette smoking associated with a chronic cough. He denied any systemic symptoms or physical changes. Bronchoscopy disclosed normal airways and bronchial washings were nondiagnostic.

A wedge resection of the left lung mass and multiple biopsies of the mediastinal lymph nodes revealed granulomatous disease. The lymph nodes were fibrotic in consistency, with no evidence of any calcification. These nodes extended throughout the mediastinum including the aortopulmonary window. The Gomori's methenamine-silver stain showed budding yeast forms consistent with histoplasma (Fig 3). The patient made an uneventful recovery from the operative procedure.

Blood cultures and a bone marrow biopsy showed no evidence of disseminated histoplasmosis. Skin testing was positive for immunologic reactivity to histoplasma. The patient was given oral ketoconazole (400 mg/day). Treatment was continued for 12 months. The patient showed no clinically significant change in his chest roentgenogram or in his hoarseness more than 1 year after operation. He is otherwise asymptomatic and leading an uncompromised lifestyle.

Comment

Disseminated histoplasmosis classically is seen as a mild chronic disease that may be associated with weight loss, fever, fatigue, and multiorgan involvement [2]. A common finding of the disease is an oropharyngeal granulomatous ulceration causing local symptoms including hoarseness. Previous reports of hoarseness in histoplasmosis described lesions in the oropharynx, but not vocal cord paralysis [3].

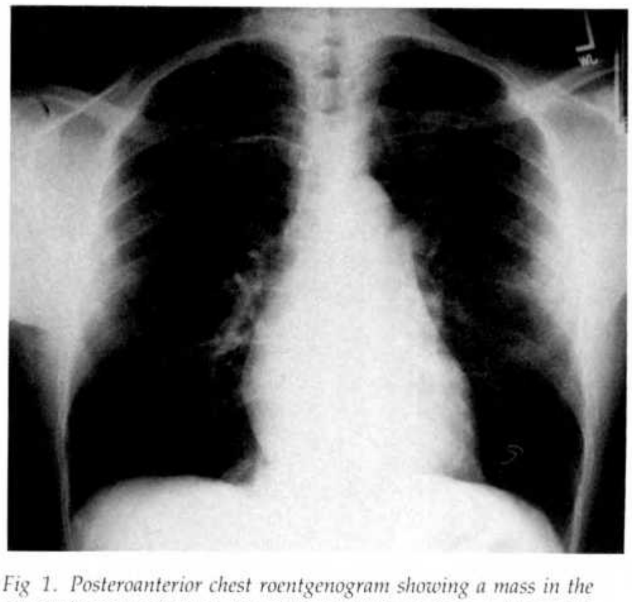

Fig 1. Posteroanterior chest roentgenogram showing a mass in the aortopulmonary window. A peripheral left lung mass is obscured by the scapula.

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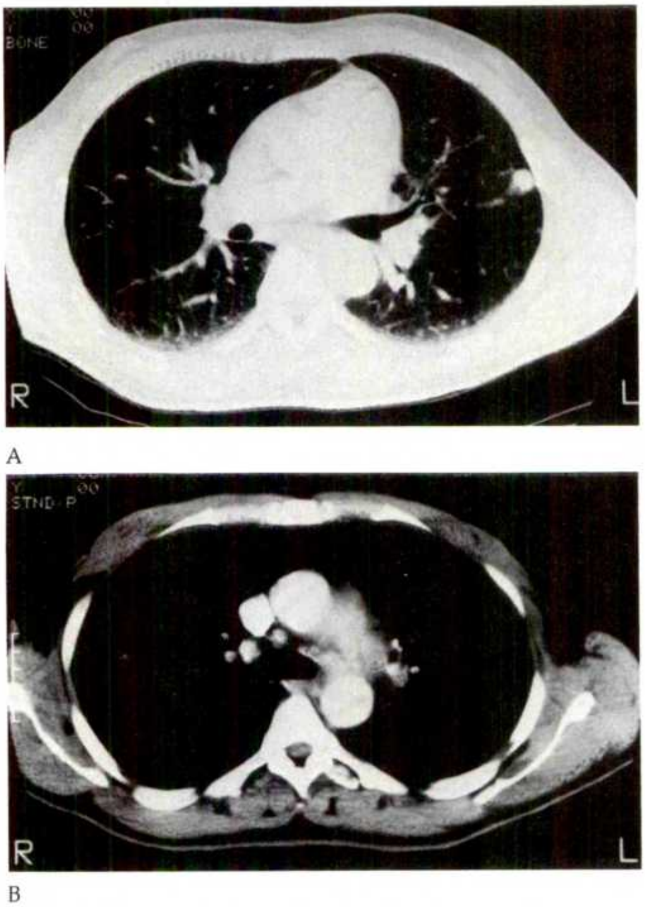

Fig 2. (A) Computed tomographic scan of the chest showing a mass in the aortopulmonary window. (B) Computed tomographic scan with a 1¼ × 1-cm noncalcified mass in the left upper lobe of the lung.

Hypersensitivity to the antigens of *H capsulatum* causes an intense inflammatory response resulting in two types of mediastinal fibrosis. The first shows a healing focus of infection with external layers of collagen and fibroblasts. The enlarging fibrotic capsule surrounds and destroys adjacent normal tissue in the mediastinum. Commonly affected are the superior vena cava, main bronchi, pulmonary veins, and pulmonary arteries. Symptoms are related to the involvement of these structures [4–6]. The lymphadenopathy of the fibrotic reaction may be mistaken radiologically for a neoplasm [7, 8]. The second response has unusually large caseous lymph nodes surrounded by a thin fibrotic capsule forming a mediastinal granuloma. This latter response causes symptoms much less frequently than the mediastinal fibrotic type of reaction. Previous reviews have lacked any cases in which recurrent laryngeal nerve paralysis developed secondary to mediastinal fibrosis or granuloma formation.

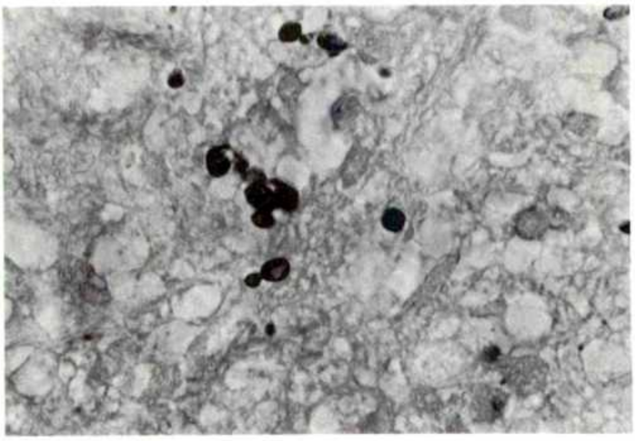

Fig 3. Photomicrograph of the lung specimen demonstrating histoplasma organisms, one with bud. (Oil immersion, Gomori's methenamine-silver, ×400 before 50% reduction.)

Mediastinal adenopathy, a pulmonary nodule, and recurrent laryngeal nerve injury make differentiation between a benign or malignant process difficult. Tissue sampling is necessary to establish a diagnosis [5]. Definitive diagnosis in this case was made by thoracotomy.

Although the potential exists for recurrent laryngeal nerve paralysis by the mediastinal fibrosis of histoplasmosis, we could find no previous reports of such a case. The presence of this symptom in a patient with mediastinal fibrosing histoplasmosis and a peripheral granulomatous lung mass indicates the ability of this infectious disease to mimic a malignant process.

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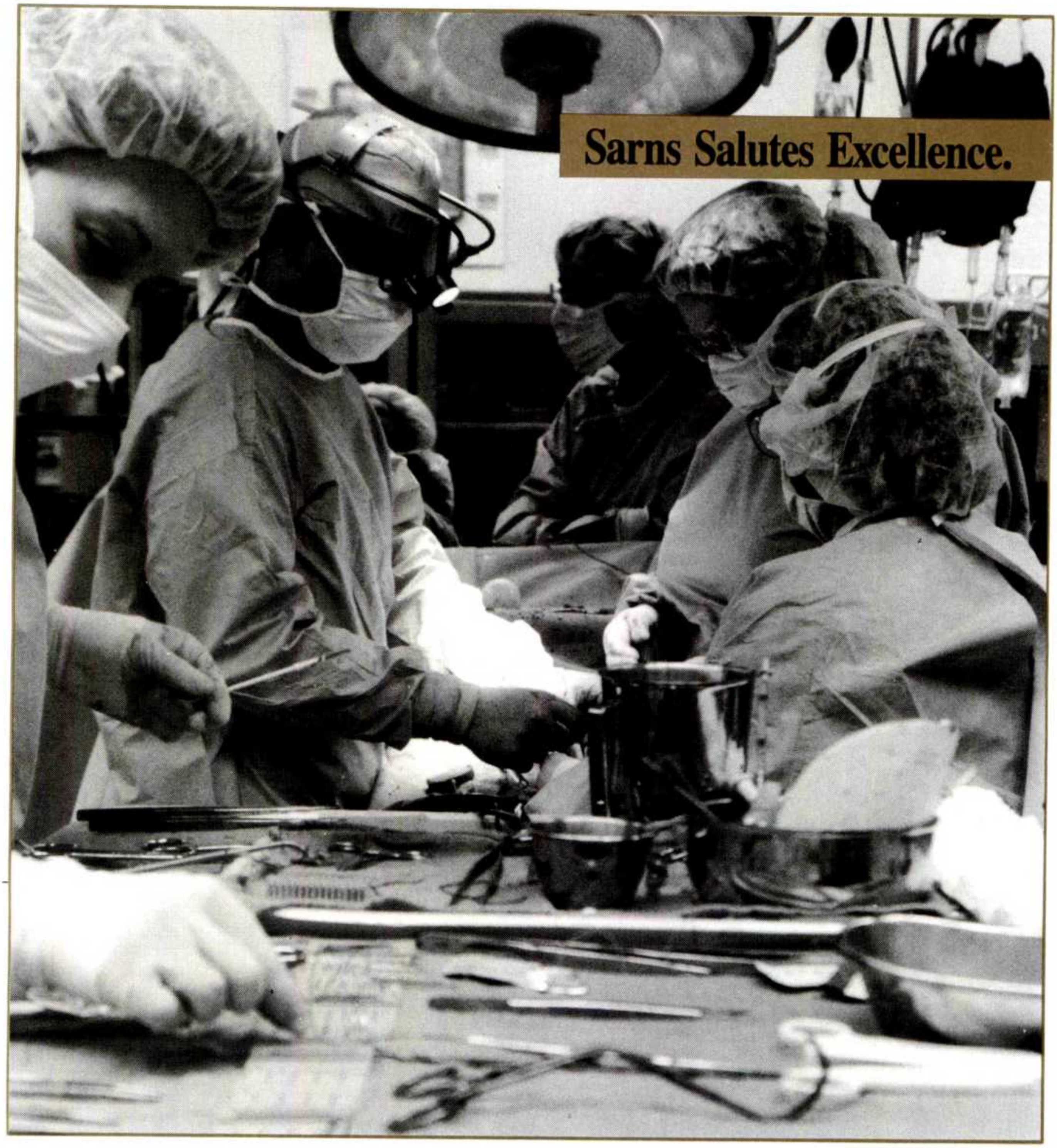
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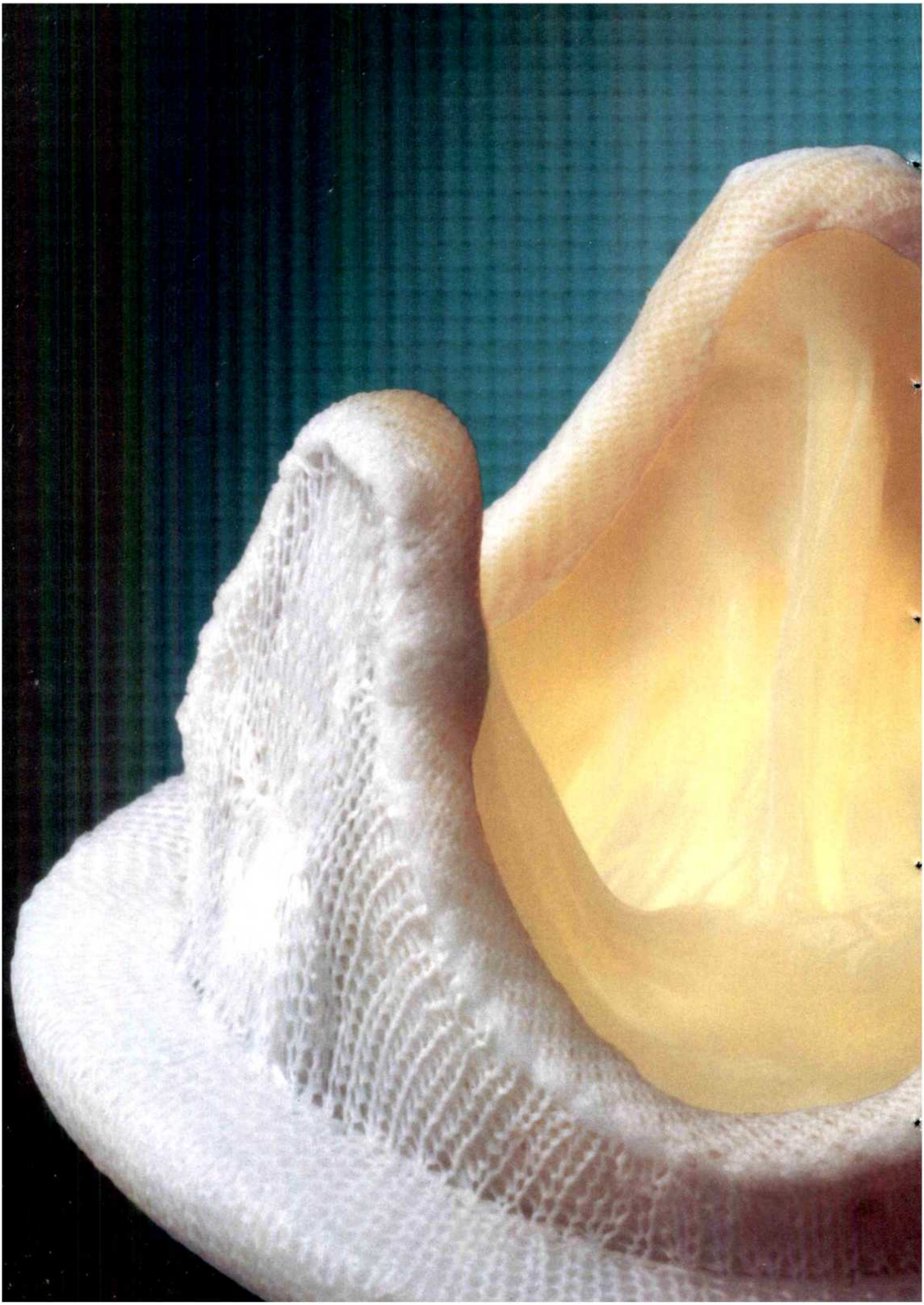

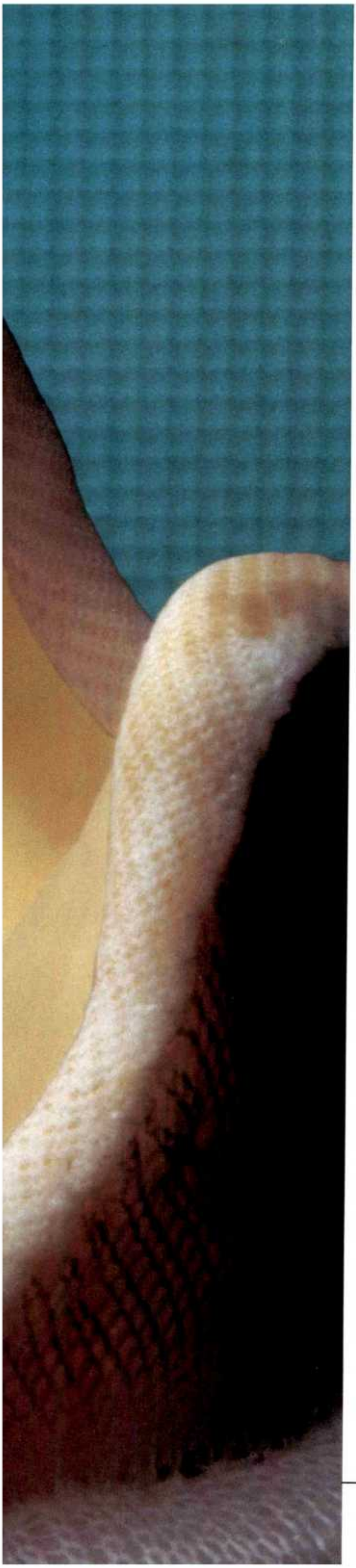

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The decision to use a tissue valve must ultimately be made by the physician on an individual basis after a careful evaluation of the short- and long-term risks and benefits to the patient are made, and alternative methods of treatment are considered. Overall durability, especially long-term, has not been established for bioprostheses. Serious complications, sometimes leading to replacement of the valve and/or death, may be associated with the use of prosthetic valves (see Complications). A full explanation of the benefits and risks should be given to each prospective patient before surgery. **Note:** Bioprostheses should be used with caution in the presence of severe systemic hypertension or when the anticipated patient longevity is longer than the known longevity of the prosthesis. Careful and continuous medical follow-up (at least by an annual visit to the physician) is advised so that valve-related complications, particularly those related to material failure, can be diagnosed and properly managed. Recipients of prosthetic heart valves who are undergoing dental procedures should receive prophylactic antibiotic therapy to minimize the possibility of prosthetic infection. Bioprosthetic heart valve recipients should be maintained on anticoagulant therapy, except where contraindicated, during the initial healing stages after implantation, approximately 2 to 3 months. Anticoagulants should then be discontinued over a period of 10 days, except in those patients for whom indefinite anticoagulant protection is indicated, i.e., in the absence of sinus rhythm and in patients with a dilated left atrium, calcification of the atrial wall or history of previous atrial thrombosis. The appropriate anticoagulation therapy must be determined by the physician on an individual basis. **Adequate rinsing with physiological saline, as described in the Technique section, is mandatory before implantation to reduce the glutaraldehyde concentration. No other solutions, drugs, chemicals, antibiotics, etc. should ever be added to the glutaraldehyde or rinse solutions, as irreparable damage to the leaflet tissue, which may not be apparent under visual inspection, may result. The Carpentier-Edwards bioprostheses must be kept moist at all times. Drying out will cause irreversible damage to the leaflet tissue. To prevent drying out during implantation, the valves should be irrigated periodically on both sides with sterile physiological saline.** Passing diagnostic catheters and transvenous pacing leads across a Carpentier-Edwards bioprosthesis could damage the valve. Consequently, it is recommended that these procedures be avoided. Both the valve and glutaraldehyde storage solution are **sterile.** The outside of the jar is **not sterile** and must **not** be placed in the sterile field. Furthermore, the valve and container should **never** be subjected to sterilization procedures involving ethylene oxide, propylene oxide, steam, or irradiation. Storage between 10°C and 25°C is recommended; however, care should be exercised to avoid freezing or extreme heat, which may damage the valvular tissue. **Any valve that has been frozen or exposed to temperatures greater than 60°C should not be used for human implantation.** Glutaraldehyde may cause irritation of the skin, eyes, nose, and throat, and may also cause skin sensitization. Avoid prolonged or repeated contact or prolonged breathing of the vapor. Use only with adequate ventilation. In the event of contact, immediately flush the affected area with water. In the event of contact with eyes, seek medical attention. **Surgical Precautions: 1.** When choosing a valve for a given patient, the size, age, and physical condition of the patient in relation to the size of the prosthesis must be taken into consideration to minimize the possibility of obtaining a suboptimal hemodynamic result. The selection of a valve, however, must ultimately be made by the physician on an individual basis after carefully weighing all of the risks and benefits to the patient. **2.** Due to the relative flexibility of the frame, care must be exercised to avoid the use of too large a prosthesis to prevent folding or extreme deformation of the stent that may render the valve incompetent. Oversizing may create highly localized mechanical stresses resulting in tissue failure in the form of detachment of the leaflets or porcine aortic wall from the valve stent posts. **3.** Left ventricular perforation or ulceration has been reported. Both open and closed chest cardiac massage in patients with an open strut mitral prosthesis increases the risk of ventricular perforation. Additionally, lifting, flexing, and manual compression of the distended heart after discontinuation of bypass may cause a commissure strut to be pushed through the endocardium, producing a dissecting hematoma with subsequent uncontrolled hemorrhage. **4.** The spacing of the sutures in the remnant of the valvular orifice and the prosthesis suture ring must be carefully matched to avoid folding of the leaflets or extreme distortion of the orifice. Edwards CVS Division has received reports in which individual mattress sutures, spanning a distance of 10 to 15mm, produced a purse-string effect causing compression of the valve orifice. **5.** When using interrupted sutures, it is important to cut the sutures close to the knots and to ensure that exposed suture tails will not come into contact with the leaflet tissue. Cases have been reported in which bioprostheses developed severe regurgitation and had to be replaced as a result of wear due to contact with sutures. **6.** Unlike rigid mechanical valves, the stent wall is soft and will not resist needle penetration. Accordingly, extreme care must be exercised when placing sutures through the sewing margin to avoid penetration of the side wall of the stent and possible laceration of the leaflet tissue. **7.** As with all prostheses that have open cages, free struts, or commissure supports, care must be exercised, particularly in mitral replacement, to avoid looping or catching a suture around the free legs which would interfere with proper valvular function. **8.** The stent of the bioprosthesis is asymmetrical to conform to the natural shape of the porcine aortic valve. Consequently, the commissure supports are not equidistantly spaced. The contrasting suture markers in the sewing ring of the mitral valve denote the greatest intercommissural distance. These two commissure supports should be oriented so as to straddle the ventricular outflow tract to avoid murmurs due to blood flow turbulence. **Note:** The contrasting suture material should not be removed from the prosthesis. **9.** In the aortic position, the struts should correspond to the remnants of the natural commissures so as not to obstruct the coronary ostia. **10.** A serial number tag is attached to the sewing ring of each valve by a suture. This serial number should be checked against the number on the jar; if any difference is noted, the valve should be returned unused. This tag should not be detached from the valve until implant is imminent. Care should be exercised to avoid cutting or tearing the suture ring cloth during removal. **11.** Gentle handling is required for all implantable devices. If the valve is dropped, damaged, or mishandled in any way, it must not be used for human implantation. **Complications:** As with all prosthetic heart valves, serious complications, sometimes leading to death, may be associated with the use of tissue valves. In addition, complications due to individual patient reaction to an implanted device, or to physical or chemical changes in the components, particularly those of biological origin, may occur at varying intervals (hours or days) necessitating reoperation and replacement of the prosthetic device. Complications associated with the use of Carpentier-Edwards bioprostheses include stenosis, regurgitation through an incompetent valve, perivalvular leak, endocarditis, hemolysis, thromboembolism, thrombotic obstruction, bleeding diatheses related to the use of anticoagulant therapy, ventricular perforation by stent posts, looped sutures, and malfunctions of the valve due to distortion at implant or physical or chemical deterioration of valve components. Types of tissue deterioration include infection, calcification, thickening, perforation, suture abrasion, instrument trauma, and leaflet or aortic wall detachment from the valve stent posts. These complications may present clinically as abnormal heart murmur, shortness of breath, exercise intolerance, dyspnea, orthopnea, anemia, fever, arrythmia, hemorrhage, transient ischemic attack, stroke, paralysis, low cardiac output, pulmonary edema, congestive heart failure, cardiac failure, and myocardial infarct. **Note:** Based on reports in the literature on tissue valves, there appears to be an increased incidence of leaflet calcification in patients under the age of 20. In this regard, animal research studies show that a high systemic calcium level can lead to early calcification. Furthermore, at least one published report describes an apparent causal relationship between the consumption of daily calcium supplements and early leaflet calcification in an adult. When feasible, repeated intravenous injections containing calcium should be avoided during the postoperative period; and excessive milk or dairy product consumption should be avoided in children. There are no clinical data presently available demonstrating increased resistance of Carpentier-Edwards bioprostheses to calcification as compared to other commercially available porcine valves. **Caution:** Federal (USA) law restricts this device to sale by or on the order of a physician. Baxter International Inc. is the owner of the following US patent which covers the products described herein: 4,106,129. Patent numbers in foreign countries supplied upon request. See package insert accompanying product for a full discussion of warnings, contraindications, complications and prescribing information. Carpentier-Edwards® is a registered trademark of Baxter International Inc.

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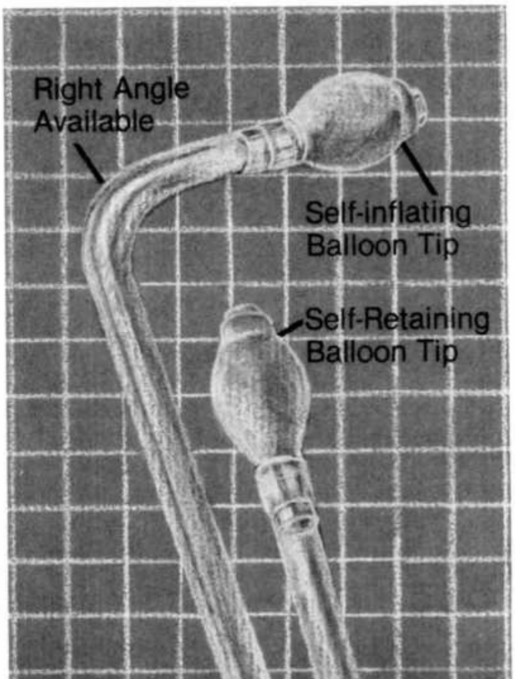
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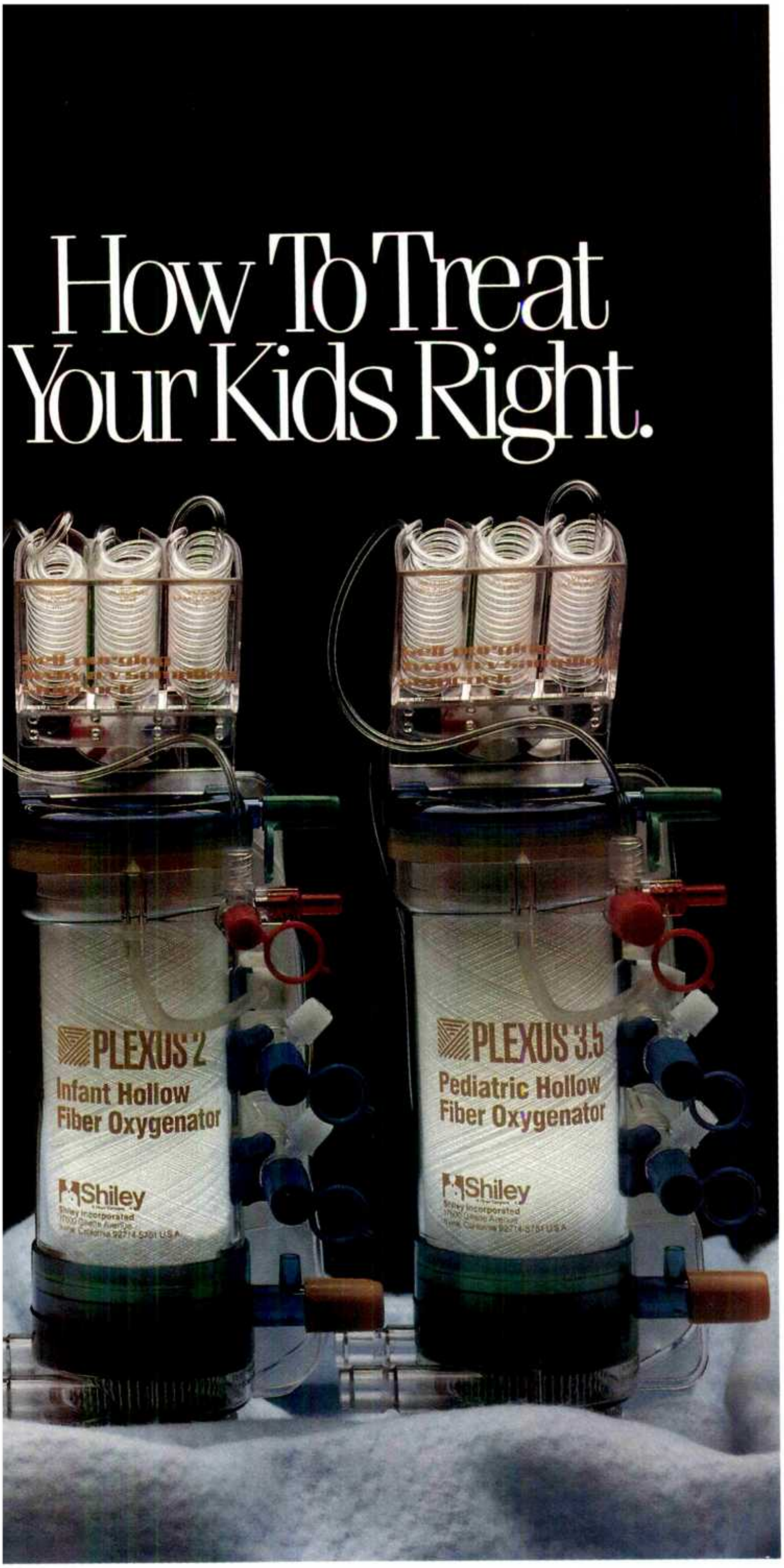
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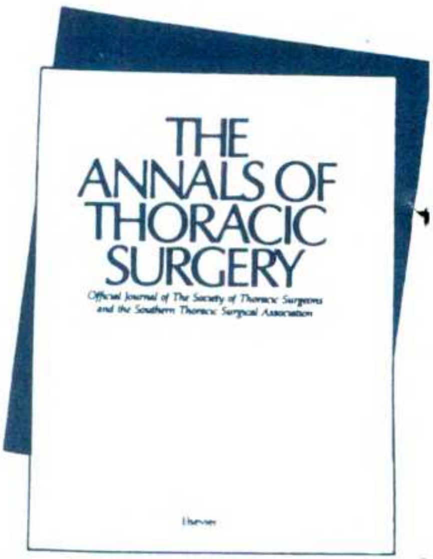
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Malignant Thymoma Presenting as Intracardiac Tumor and Superior Vena Caval Obstruction

B. Airan, MCh, R. Sharma, MCh, K. S. Iyer, MCh, P. K. Kalia, MD, M. K. Singh, MD,
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A case of malignant thymoma with intracardiac infiltration and intrinsic superior vena caval obstruction due to the tumor is reported. Intracardiac invasion by a thymoma is rare and for this reason is believed worthy of a report.

(Ann Thorac Surg 1990;50:989–91)

A 38-year-old man had a 6-month history of right hypochondrium pain, abdominal distention, and weight loss. Examination revealed engorged, nonpulsatile neck veins, dilated tortuous veins over the trunk with blood flow from above downward, moderate ascites, and a firm 6-cm liver. Mild icterus was present. Cardiovascular examination was normal. Chest roentgenogram showed bilateral pleural effusion with no cardiomegaly or pericardial calcification. Liver function tests were mildly deranged. Echocardiography (Fig 1A) of the heart revealed a large immobile mass measuring 9.0 × 6.0 cm occupying the right atrium and right ventricle. Inferior vena cava was free of tumor. Ultrasound examination of the liver and kidneys was normal.

In view of his poor general condition, the patient was operated on without any further investigations with the presumptive diagnosis of right atrial myxoma with bicaval obstruction.

A median sternotomy was performed. The thymus was nodular and gelatinous in appearance. A frozen section biopsy of thymus was reported as spindle-cell soft tissue sarcoma. The pericardium was opened and found adherent to the right atrium over an area approximately 2.0 cm in diameter. The right atrium, right ventricle, and superior vena cava were distended and felt solid. The inferior vena cava was tense and enormously dilated but compressible. The pulmonary artery was collapsed.

Under cardiopulmonary bypass, using ascending aortic cannulation and a single venous cannula inserted at the right atrium–inferior vena caval junction, the heart was arrested at 28°C and cardioplegia was given through the aortic root. The right atrium was opened widely. A large tumor of variegated and nodular appearance was found that seemed to be attached to the entire right atrial endocardium. It extended across the tricuspid orifice into the right ventricle. The anterior leaflet of the tricuspid valve was grossly involved and the septal leaflet was found embedded in the tumor mass. Right ventricular extension was limited to the body alone. The superior vena cava had no lumen because of tumor ingrowth and could not be cleared. The right atrium and right ventricle were cleared of tumor by removing it piecemeal (Fig 2). After the right atrium was closed, the patient was weaned off cardiopulmonary bypass.

The patient made an uneventful recovery: The ascites disappeared, the liver regressed to normal size, and the pleural fluid did not collect again after initial drainage of the bilateral pleural effusions. He continued to have prominent neck veins draining through tortuous trunk veins downward into the inferior vena cava.

The histopathological studies were reviewed by Dr Raffaele Lattes and we herewith reproduce his report in its entirety:

> The tumor is highly cellular and consists of polygonal, elongated and rounded cells, which are arranged without any glandular or squamoid differentiation, and are associated with a moderate vascularity. The tumor cells arrange themselves sometimes in a palisading fashion around the stroma surrounding the blood vessels. The blood vessels are occasionally surrounded by an empty space (probably lymphatic) which is characteristic of tumors of the thymus. Mitotic activity is low (Fig 3).
>
> Reticulin stains show a paucity of reticulin fibrils which is very much in favour of an epithelial type of tumor. There is a sprinkling of lymphocytes. *PAS stains* [periodic acid-Schiff] show no PAS positive material in the cytoplasm of the tumor cells and they emphasize the "epithelial" pattern. *Stains for EMA* (epithelial membrane antigen) are positive in numerous tumor cells, especially those that are arranged in a row around the stroma of the trabeculae. *Stains for keratin* are weakly positive in the tumor cells. *Stains for CLA* (common leukocyte antigen) are negative in the tumor cells, but positive in the lymphoid cells scattered through the neoplastic tissue. *Stains for CEA* [carcinoembryonic antigen] are negative. *Immunostains* for lymphocytes type show that the scattered lymphocytes are T cells (positive for L.22, negative for L.26).
>
> The histological and immunohistochemical findings confirm the diagnosis of thymoma.

Postoperative echocardiography showed right atrium and right ventricle free of tumor with superior vena cava still containing tumor (Fig 1B). A thoracic computed tomographic scan showed tumor in the right lobe of the thymus extending to the right lung laterally, extending to the aortic arch medially, and compressing the tracheal bifurcation posteriorly. The superior vena cava was

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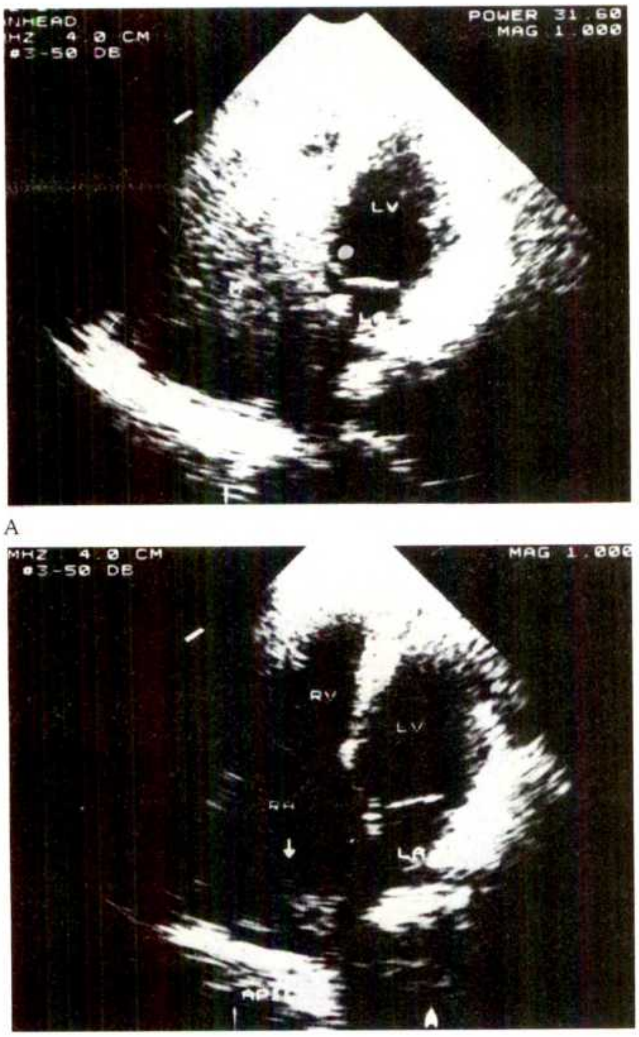

Fig 1. (A) Preoperative two-dimensional echocardiogram showing the intracardiac mass (M) occupying the right atrium and right ventricle. (B) Postoperative echocardiogram showing right atrium and right ventricle free of tumor. (LA = *left atrium;* LV = *left ventricle;* RA = *right atrium;* RV = *right ventricle.)*

thrombosed and the right atrium and right ventricle were free of tumor. The patient was sent for radiotherapy.

Comment

Thymoma, the most common tumor of the anterior mediastinum, is malignant in 30% to 50% of cases [1, 2]. Diagnosis of malignant thymoma is based on gross appearance of the tumor and features of surrounding invasion rather than on histology [1–4]. Local invasion occurs most often into the pleura (30%), pericardium (25%), lung (8%), recurrent laryngeal nerve (4%), and rarely the great vessels [5]. Twenty percent of malignant thymomas ultimately spread to the pleural surfaces by implantation; in only 5% are there extrathoracic metastases. Thymomas are known for their frequent association with myasthenia gravis (25%) as well as the other, more uncommon parathymic syndromes (eg, pure red cell aplasia and hypogammaglobulinemia) [1]. The present case had none of these associations. Superior vena cava obstruction is a

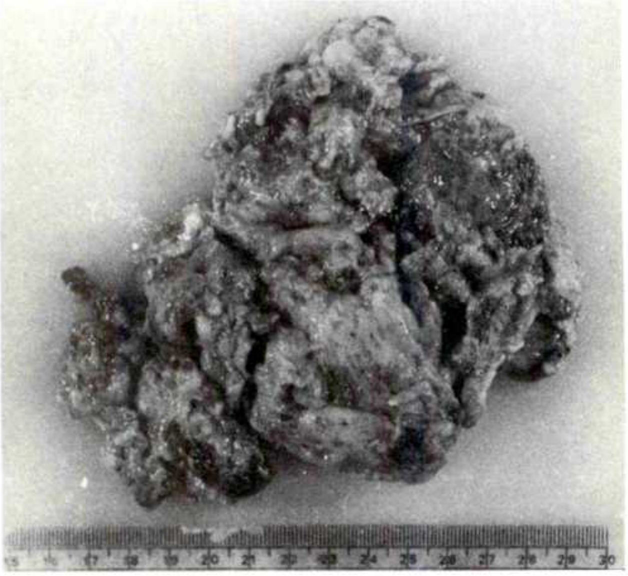

Fig 2. Excised intracardiac mass.

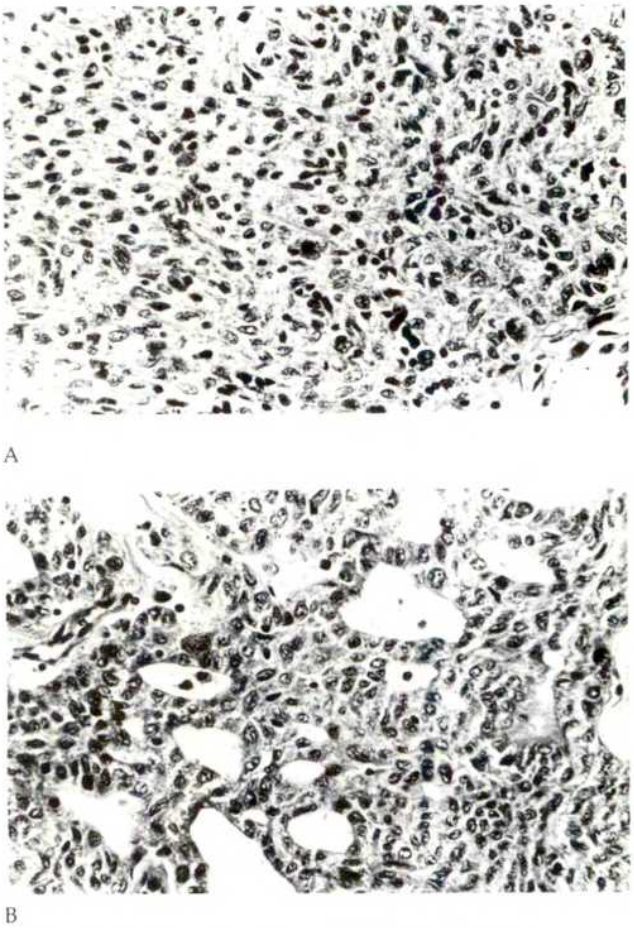

Fig 3. Histological sections of the excised intracardiac mass.

presenting feature of thymoma in less than 2% of cases [2]. Superior vena caval obstruction, when it occurs, is more by extrinsic compression than by infiltration into the lumen. Although pericardial involvement is well documented, extension of the tumor into the cardiac chambers is virtually unknown. Only a single report of right atrial extension of a thymoma exists in the world literature [6]. Four primary pericardial thymomas have been described by McAllister and Fenoglio [7] out of a total of 444 tumors of heart and pericardium in their series, but none had cardiac extension.

Histologically thymic malignancy may be composed of (1) predominantly spindle cells, (2) predominantly lymphocytes, (3) differentiated epithelial cells, or (4) undifferentiated sarcomatoid epithelial cells [4]. The present case probably belongs to the first category. Treatment of malignant thymomas is radical extirpation followed by adjuvant or combination radiotherapy with or without chemotherapy [5, 8]. As we were initially misled by the frozen section report, radical extirpation of the tumor was not accomplished. Later, computed tomographic scan demonstrated a huge unresectable tumor extending from the right lung to the base of the heart medially and to the tracheal bifurcation posteriorly. Reoperation was, therefore, not attempted and the patient was referred for radical radiotherapy for his residual tumor.

We are extremely grateful to Dr Raffaele Lattes for reviewing the histopathological slides.

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Job's Syndrome: A Rare Cause of Recurrent Lung Abscess in Childhood

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A clinical syndrome characterized by recurrent staphylococcal infection of the skin and respiratory tract from birth was described in 1966 and referred to as Job's syndrome. Marked hyperimmunoglobulinemia E was later found to be associated with this syndrome. This article describes a case of Job's syndrome as a cause of recurrent lung abscess during childhood necessitating lung resection.

(Ann Thorac Surg 1990;50:992–4)

An 11-year-old boy was referred to the general thoracic surgery service in 1989 for assessment of recurrent staphylococcal lung abscesses. History revealed that at age 8 months he had an episode of pneumonia of the right lung, which evolved to empyema (Fig 1). The culture from thoracentesis identified *Staphylococcus aureus* as the offending organism. The patient was treated with intravenous administration of an antistaphylococcal antibiotic and drainage by closed tube thoracostomy with complete resolution of the empyema.

During the succeeding 10 years, the patient had numerous episodes of bronchitis, otitis media, and subcutaneous abscesses of the face, neck, and back, which were not accompanied by fever and erythema or warmth of the abscess. He was allergic to a wide variety of environmental agents including soap, fur, fuzzy toys, dust, and feathers. Neither his parents nor his 3 siblings had the same problem.

In January 1987 at the age of 9 years, the patient had a high fever with chill, nausea, emesis, generalized malaise, fatigue, dyspnea, and a productive cough. Cultures of sputum samples showed heavy growth of *S aureus*. Chest roentgenograms revealed a large cavity of the right lower lobe with an air-fluid level. The lung abscess resolved with 2 weeks of high-dose intravenous oxacillin and vigorous chest physical therapy. A similar episode occurred in November 1988 and resolved with the same treatment (Fig 2).

The patient underwent a battery of immunological studies in April 1989. Major findings included a normal result for the nitroblue tetrazolium reduction test after phagocytosis, thus suggesting normal leukocyte chemotaxis, and a markedly elevated serum immunoglobulin E level of 19,200 μg/L (8,000 IU/mL) (normal, 0 to 240 μg/L [100 IU/mL]). Based on the clinical history and the elevated level of immunoglobulin E, the diagnosis of Job's syndrome was made. Computed tomography of the chest showed extensive parenchymal destruction of the right lower lobe with a large residual cavity and marked bronchiectasis.

Admission physical examination revealed a boy with dark brown hair and normal growth for his age (149 cm tall and weighing 48 kg). There were multiple skin scars over the face, neck, back, and buttocks. There was no evidence of acute otitis media or externa, or sinusitis. There was no palpable cervical, axillary, or inguinal lymphadenopathy. The liver and spleen were not enlarged. Breath sounds over the right lower lung were markedly diminished. The remaining examination was essentially normal.

Admission white blood cell count was 8×10^6/L (8,000/mL) with 45.6% each of neutrophils and lymphocytes, 8.8% monocytes, and no eosinophils. Hemoglobin and hematocrit were 123 g/L and 0.37, respectively, with normal indices. Serum electrolytes were normal. The 12-channel chemical profile was remarkable only for an elevated level of alkaline phosphatase (190 U/L; normal, 20 to 94 U/L). On room air, arterial oxygen tension and arterial carbon dioxide tension were 89 mm Hg and 38 mm Hg, respectively, with 99% hemoglobin saturation. Chest roentgenography revealed a large pneumatocele of the right lower lobe without an air-fluid level (Fig 3).

Because of the extensive destruction of the right lower lobe, which clearly was the nidus of the recurrent lung abscesses, an elective right lower lobectomy was recommended. The patient underwent an uneventful right posterolateral thoracotomy with right lower lobectomy despite extensive pleural adhesion and fibrosis. His postoperative course was quite unremarkable. At the 4-month follow-up, he was at the level of his preoperative exercise tolerance without further interval pneumonia. Final pathological review of the right lower lobe showed an abscess cavity with aspergilloma, follicular and saccular bronchiectasis, and acute pneumonia with focal pulmonary adenomatosis.

Comment

In 1966, Davis and colleagues [1] described a clinical syndrome in 2 unrelated girls with red hair and fair skin who, from birth, experienced chronic eczema, recurrent

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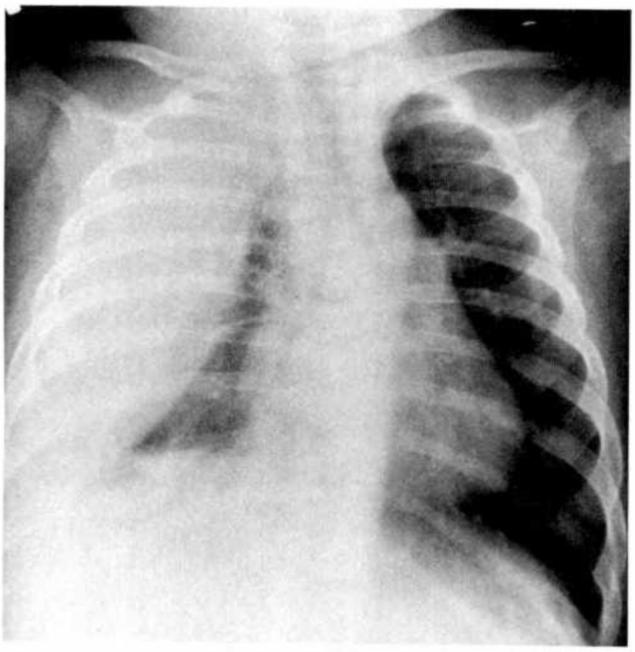


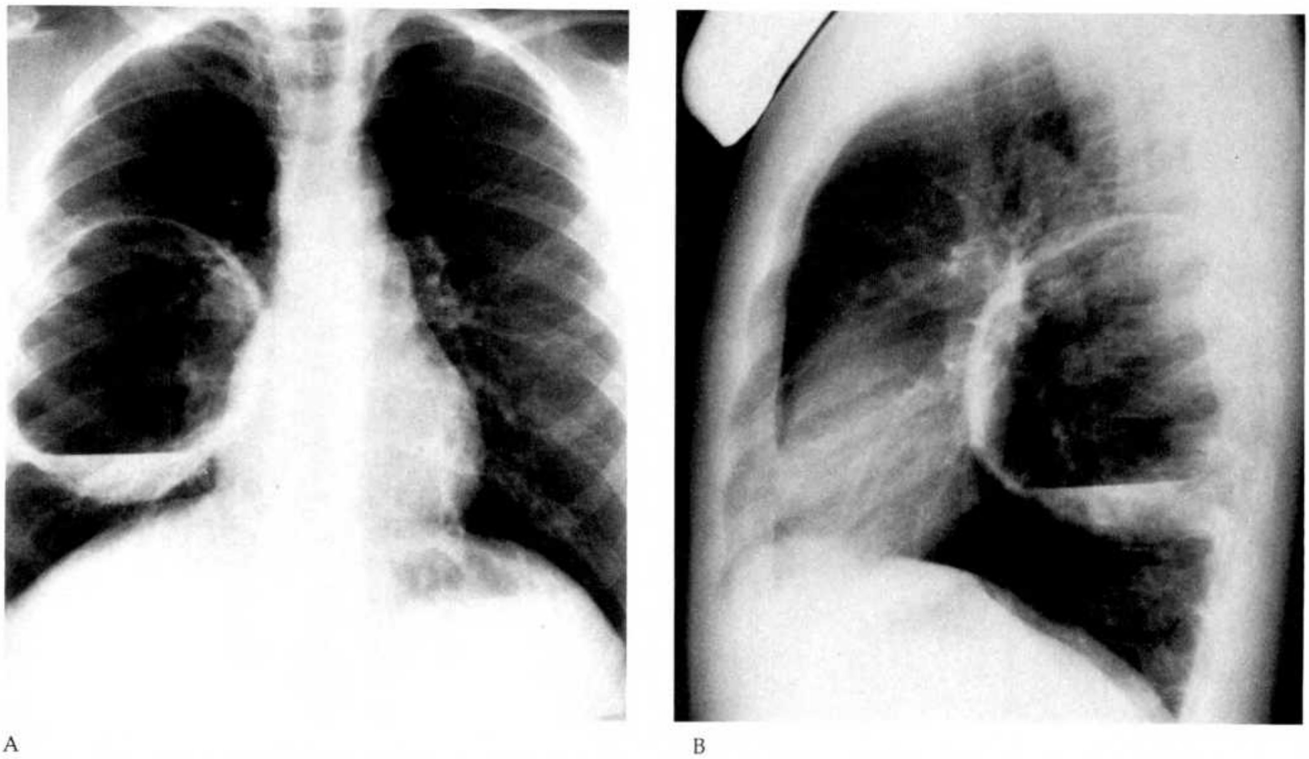
Fig 1. Anteroposterior chest roentgenogram made when the patient was 8 months old shows right-sided empyema.

staphylococcal infections of the sinopulmonary tract, and an abnormal inflammatory response with the formation of "cold" staphylococcal abscesses. Davis and colleagues referred to this syndrome as Job's syndrome because of its resemblance to the affliction of the biblical figure. In 1972, Buckley and associates [2] described hyperimmunoglobulinemia E in 2 boys with a similar clinical syndrome. Two years later, Hill and co-authors [3] reported hyperimmunoglobulinemia E and defective chemotaxis of polymorphonuclear leukocytes in the 2 original girls with Job's syndrome.

Patients with Job's syndrome generally show a wide range of symptoms during the first few months of life. These symptoms include the characteristic cold staphylococcal abscesses, chronic otitis media and externa, eosinophilia, mucocutaneous candidiasis, recurrent bronchitis, and staphylococcal pneumonia.

The immunopathogenesis of the recurrent staphylococcal infection in patients with Job's syndrome remains unknown. A primary defect in the function of polymorphonuclear leukocytes as evaluated by chemotaxis is not always demonstrated in these patients. Other observed immunological abnormalities include the presence of anti–*S aureus* and anti–*Candida albicans* immunoglobulin E, deficient anti–*S aureus* immunoglobulin A, and elevated serum total immunoglobulin D. In addition, deficiencies of delayed hypersensitivity have also been noted. Patients with Job's syndrome also seem to be predisposed to unusual fungal infections such as cryptococcosis and histoplasmosis of the esophagus [4] and the colon [5]. A genetic basis for this syndrome has been suggested, as recurrent infection and hyperimmunoglobulinemia E have been identified in several family members [6] and in a newborn whose mother has Job's syndrome [7].

Pneumatocele and bronchiectasis frequently develop as



A B

Fig 2. (A) Posteroanterior and (B) lateral chest roentgenograms made in November 1988 show right lower lobe abscess with an air-fluid level.

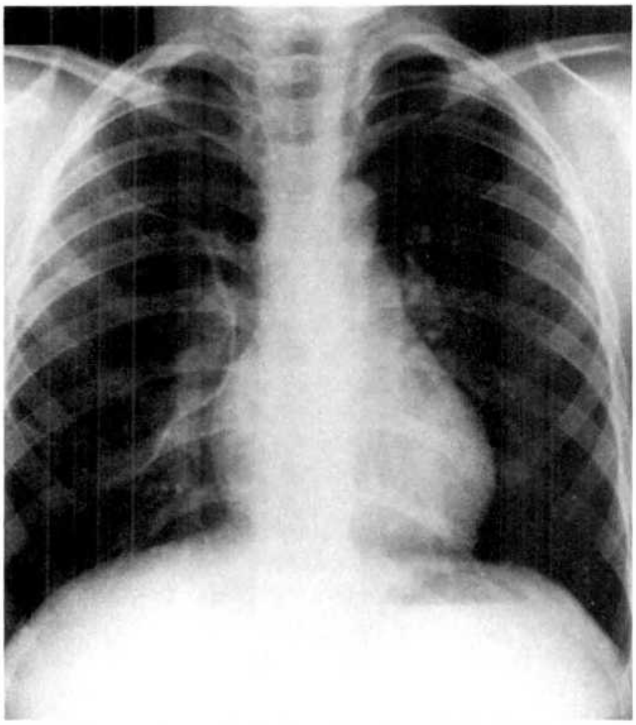


Fig 3. Posteroanterior chest roentgenogram made on admission in August 1989 reveals persistent large right lower lobe pneumatocele.

a result of repeated staphylococcal pneumonia in patients with Job's syndrome, thus predisposing them to recurrent lung abscess formation and, infrequently, massive hemoptysis. Because the prevalence and the natural history of this problem have not been documented in the literature, the role of surgical resection and its timing in the overall management of this unique group of patients have not been defined.

When assessing a child or young adult with a lifelong history of skin infections and staphylococcal pneumonia, the diagnosis of Job's syndrome should be considered along with other unusual childhood diseases such as cystic fibrosis, chronic granulomatous disease, and Kartagener's syndrome associated with congenital ciliary dysfunction. In conjunction with the pediatrician and the immunologist, the appropriate immunological workup should be performed.

Once the diagnosis of Job's syndrome has been established, the chronicity of the pulmonary suppuration should be assessed by a thorough history from the patient and the family members. The extent of pulmonary parenchymal destruction and presence of cavitation might be adequately delineated by computed tomography. The functional status of the diseased portion of the lungs should be evaluated by regional ventilation/perfusion scans. If a portion of the lungs is demonstrated to be extensively destroyed, nonfunctional, and clearly the nidus of recurrent lung abscess formation, elective resection should be recommended. Because of the unresolved underlying immunopathology, which will continue to predispose the patient to further loss of lung tissue, minimal resection compatible with complete removal of nonfunctional lung tissue should be performed whenever possible.

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Selective and Adjustable Pericardial Flap to Protect Internal Mammary Artery Grafts

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We describe the surgical technique of a localized and adjustable pericardial flap to protect internal mammary artery grafts. This flap allows selective pulmonary retraction, maintains pleural integrity, and saves most of the pericardium for later closure. This technique has proved to be simple and highly effective. We have used it in 80 patients and have not had any related complications.

(Ann Thorac Surg 1990;50:995–7)

The internal mammary artery (IMA), once completely mobilized from the thoracic wall, is usually long enough to reach comfortably the proximal and median segments of most coronary vessels. However, excessive medial IMA graft displacement by a hyperinflated lung is sometimes found in patients with chronic obstructive pulmonary disease. Even though intrapleural IMA routing is the easiest way of avoiding lung encroachment [1, 2], some concern still exists about postoperative detriment of pulmonary function in severely emphysematous patients owing to the pleurotomy and the thoracostomy tubes. Thus, whenever pleural integrity is preferred, maximizing IMA length [3–5] and limiting medial displacements of the lung by different pericardial manipulations [6, 7] are the sole alternatives to the more demanding free IMA graft. We describe here the technique of a selective and adjustable pericardial flap that, in our opinion, affords some advantages over other similar reported methods.

Technique

After a standard median sternotomy, the selected IMA is harvested extrapleurally from its bifurcation to the point where it crosses the subclavian vein, using low-powered cautery. When feasible, accidentally entered pleura is closed with continuous 4-0 or 5-0 suture material. The mediastinal pleura and fat are then widely cleared off the underlying pericardium, which is opened with an inverted T incision. The vertical stem of the T is divided into two curved branches as shown in Figure 1A. Once the distal anastomoses of the IMA and the venous grafts have been performed and while the patient is still on cardiopulmonary bypass, the lungs are manually insufflated by the anesthesiologist to reach an airway pressure of 30 to 40 mm Hg to evaluate IMA graft displacement. Whenever undue graft tension is observed, as is frequently the case with emphysematous patients, we protect the IMA graft by a pericardial flap, which is constructed as follows: the ipsilateral pericardial border is grasped with forceps, retracted medially, and put under moderate tension. The mediastinal pleura and fat are further dissected away from the outer pericardial surface toward its lateral aspect. The curved branch of the pericardial incision is then extended laterally to the desired level, usually to the atrial appendage on the left and close to the superior cavoatrial junction on the right. Further advancement of the lateral incision would give no additional advantage, as lung retraction depends mainly on flap length and level of thoracic anchorage. Direct visualization of the phrenic nerve is mandatory when going closer to the lung pedicle to avoid nerve injury. In doing so, the incision must be stopped at a distance of about 1 cm from the phrenic nerve.

A 2-cm-wide, cranially based pericardial strip is then tailored with the low-intensity coagulation mode of the cautery (Fig 1B). The length of this strip that, extended maximally, can reach the inferior transverse limb of the pericardial incision is calculated with consideration of protrusion of the lung when insufflated and the length of the IMA. The greater the lung protrusion and the shorter the IMA, the longer the pericardial flap should be.

Once prepared, the inferior free end of the pericardial strip is fixed to the endothoracic fascia and to the perichondrium in the lateral border of the IMA dissection groove at the level of the second or third costal cartilage by two separate 2-0 Tycron stitches (Fig 1C). Whenever some graft tension is still detected as the result of excessive lung impingement, the pericardial strip can be shortened by two additional, laterally placed plicating stitches (Fig 1D). Once we have verified again that the IMA is not disturbed by lung movements, the sternum is reapproximated. Attempts at suturing the contralateral pericardial edge to the remaining ipsilateral pericardium or to the overlying mediastinal fat should be done with a view to protecting the heart in case later reoperation is required. We have been able to close the inferior pericardium in every instance it was attempted (pericardium was left open in very aged patients). Special attention must be paid to detect any graft compression under the closed

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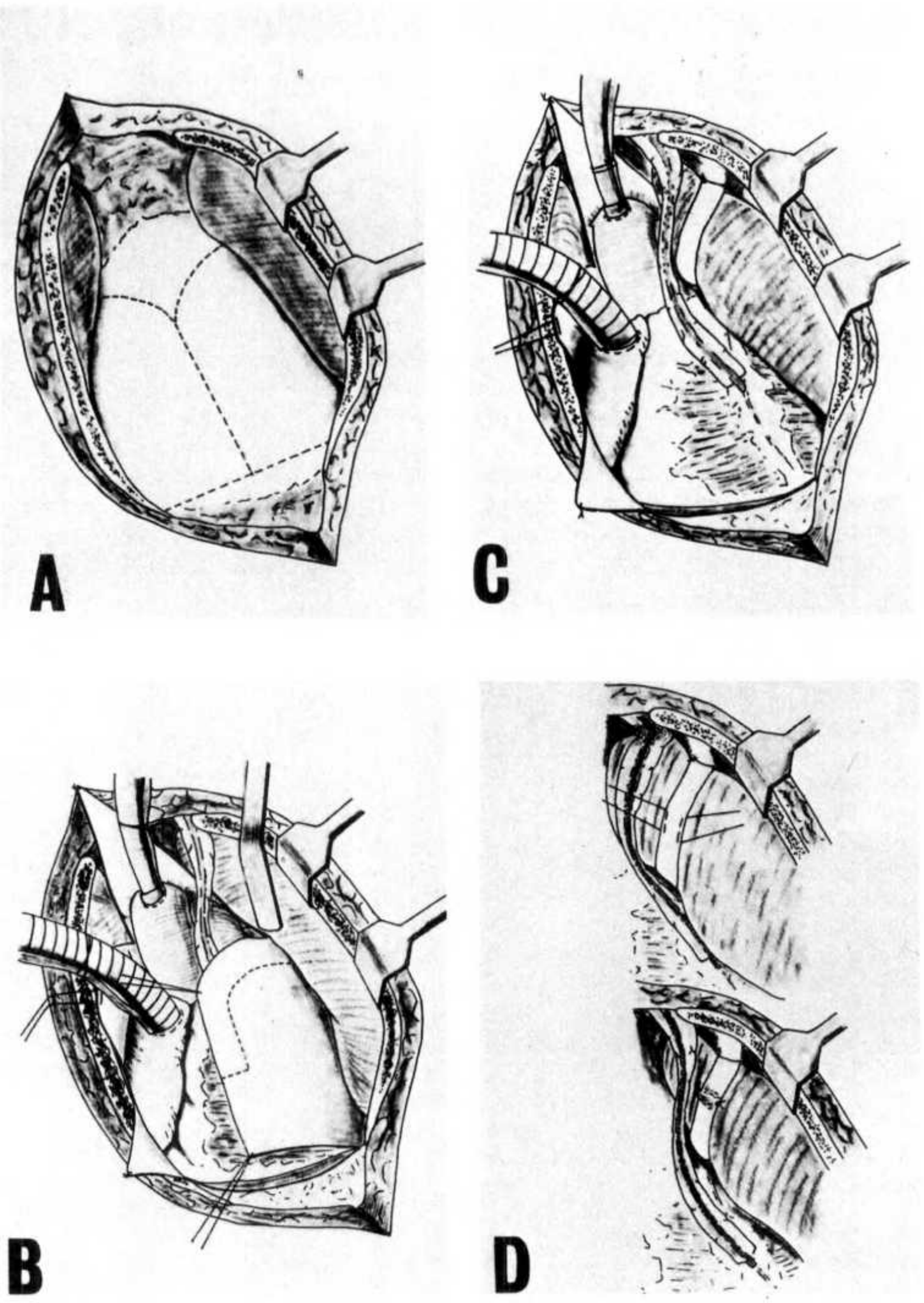

Fig 1. Technical steps in constructing and adjusting the pericardial flap. (See text for details.)

pericardium. Whenever this condition is suspected, multiple longitudinal meshlike pericardial incisions, as proposed by Milgalter and associates [8], relieve graft compression in most patients. Overall, two thirds of the patients had a satisfactory heart coverage with this technique.

Comment

Excessive stretching of in situ IMA grafts by a hyperinsufflated lung is a well-known cause of early failure. Three ways of preventing lung interference in this condition have been devised: routing the graft through the least disturbed path [1, 2], maximizing graft length [3–5], and limiting lung movements [6, 7, 9]. The first way, in which the IMA is placed medial to the upper pulmonary lobe, requires an extensive pleurotomy, as does the apical tacking technique proposed by Mills [9], to restrict lung displacements. Otherwise, whenever pleural integrity is preferred in the setting of hyperinflated lungs, the only method to prevent stretching of an IMA graft of maximal length is to limit the lung movements by a pericardial flap. Todd and colleagues [6] have recommended a wide flap constructed with all of the left pericardial border. However, this nonselective flap could result in pulmonary compression and nonprotection of the heart in case of further reoperation. These inconveniences are partially obviated with the localized flap technique proposed by Starr and Moore [7], but after using it extensively we found two disadvantages. First, the flap length was sometimes insufficient to reach the thoracic wall, thus resulting in excessive flap tension and greater postoperative discomfort. In addition, tense tacking sutures may injure the underlying lung (a bronchopleural fistula due to lung erosion by a tense suture required surgical closure in 1 of our patients). Second, misleading mediastinal widening due to lateral displacement of the pericardium may occur, especially in bilateral IMA grafting.

The main advantages of the pericardial flap described here are: (1) the greater maximal length (up to the entire pericardial height) that allows us to reach comfortably the thoracic wall in every patient; (2) the simplicity by which it is shortened, when necessary, to fit the anatomical conditions of each patient; and (3) the possibility of protecting the heart with the remaining pericardium and the mediastinal pleura and fat in view of a further operation. We have now used this technique in 80 patients, with no related problems. An unstretched graft has been achieved in every patient, even in severely emphysematous lungs. Every effort is made to close the pleura when it has been entered, although the technique may also be used in the case of extensive pleural opening. Our impression is that postoperative pain has been less than that with other techniques, and there have been no cases of mediastinal widening as most of the pericardium is left undisplaced. For all these reasons, we recommend this technique as an alternative to intrapleural IMA routing whenever maintaining pleural integrity is preferred.

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Easy Dissection of Hard and Thickened Pericardium on Constrictive Pericarditis

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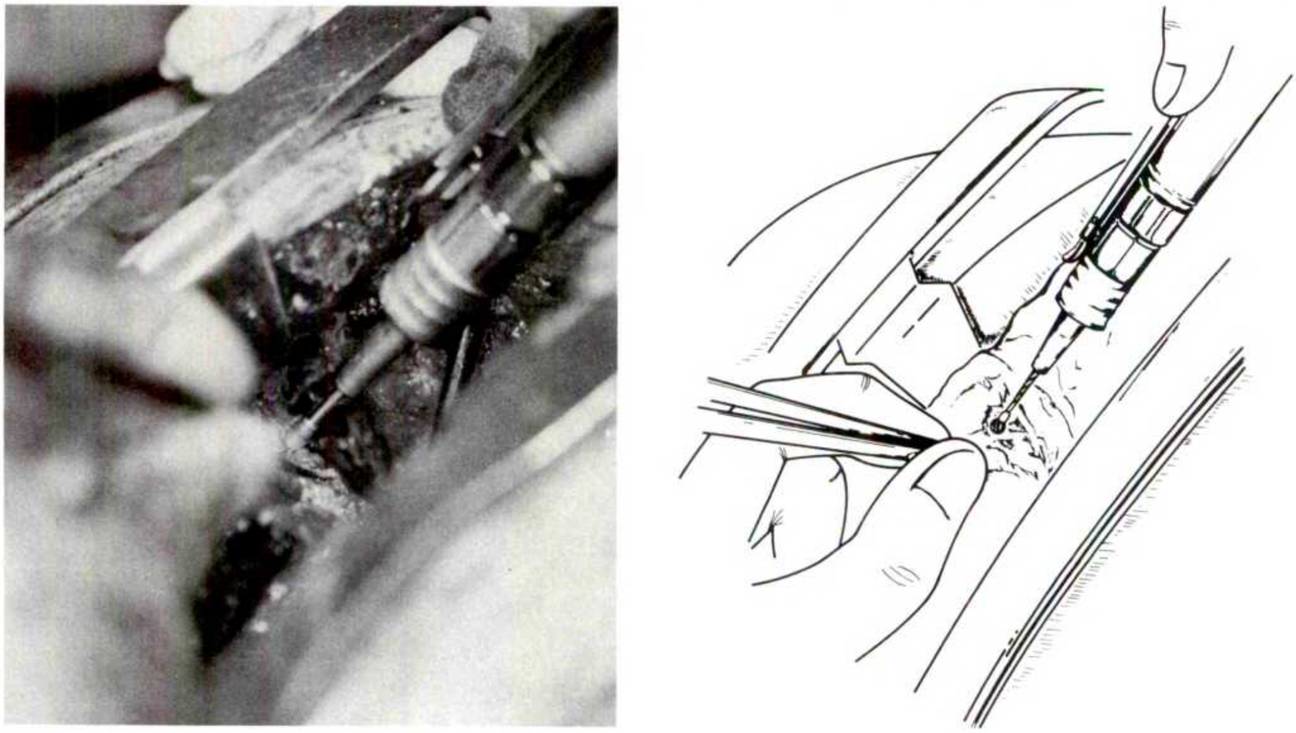
The keys to successful pericardiectomy for constrictive pericarditis are early operation and as complete a pericardiectomy as possible. With the high-speed burr it is easy and safe to dissect the calcified pericardium and define the epicardium even in a small operative field such as the inferior or posterior portion of the heart. This method has the important ability to perform very complete pericardiectomy.

(Ann Thorac Surg 1990;50:998–9)

Chronic constrictive pericarditis is a chronic inflammatory process that involves both fibrous and serous layers of the pericardium and that leads to pericardial thickness and constriction of the ventricles [1]. The symptoms of constrictive pericarditis are introduced by insufficient distention of the heart. Freedom from the constriction should theoretically reduce the symptoms.

Churchill [2] performed the first successful pericardiectomy for constrictive pericarditis in 1928. McCaughan [3], Bashi [4], and their associates reported the early and late results of pericardiectomy. McCaughan and associates [3] suggested the surgical result would continue to improve only if the diagnosis was established, and pericardiectomy should be performed before marked constriction caused myocardial damage. Bashi and co-workers also recommended early pericardiectomy for constrictive pericarditis. There is no doubt that pericardiectomy is one of the best treatments for constrictive pericarditis; however,



A

B

Fig 1. The high-speed burr was lightly touched to the surface of the constrictive pericardium. The thickened, hard pericardium was sculpted little by little up to the epicardium.

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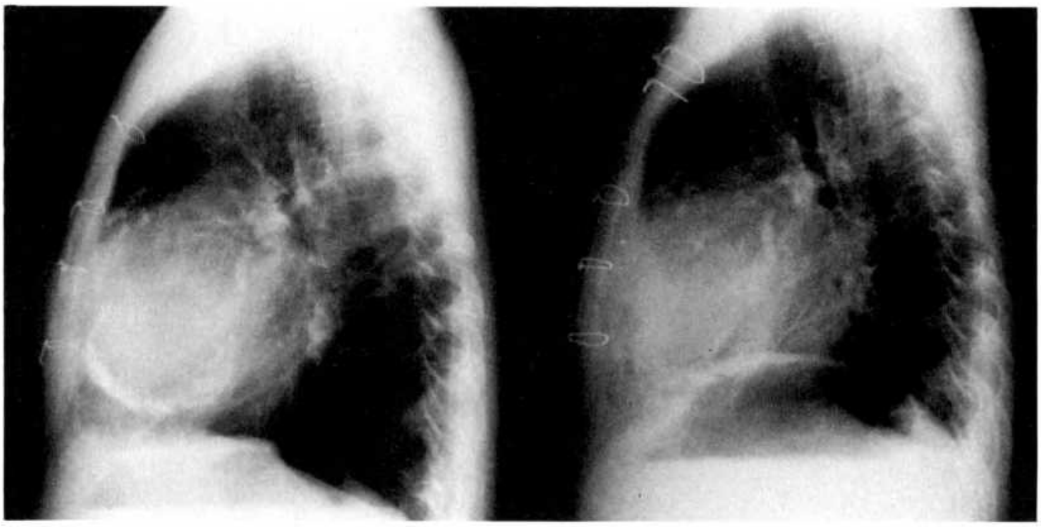

Fig 2. The postoperative chest roentgenogram (right) shows that the inferior and posterior calcified pericardium was well removed compared with that seen on the preoperative roentgenogram (left).

successful pericardiectomy depends on when and how it is performed. The high-speed burr is useful for dissecting thick and calcified pericardium.

Technique

A 44-year-old woman, who had undergone a first pericardiectomy 13 years before, underwent a second constrictive pericardiectomy by a midsternal approach. The anterior pericardial flap was dissected as far as the right side of the right atrioventricular groove and the anterior side of the left ventricle. We tried to dissect the posterior and inferior flap with scissors; however, it was impossible because the pericardium was very thick and calcified, or tightly adherent to the epicardium. The high-speed burr (Micro 100 drill, Hall Surgical Inc, Santa Barbara, CA) was used to dissect the pericardium.

The Micro 100 drill is driven by compressed dry nitrogen (99.97% pure) for cutting, drilling, and sculpting bone and enamel. The other specifications of this drill are as follows: speed, 500 to 20,000 rpm (nominal); minimum torque, 4.27 m-g (6 inch-oz); operating pressure, 11.1 kg/cm^2 (160 psi); housing diameter, 1.8 cm (0.7 inch); overall length, 15.3 cm (6 inches); and weight, 175 g (6.2 oz). This equipment has already been applied for oral, orthopedic, neurological, and plastic surgery.

The high-speed burr was lightly touched to the surface of the constrictive pericardium, which was sculpted little by little up to the epicardium (Fig 1). Some portions of the pericardium were easily destroyed with only the burr. Other parts of the pericardium, extremely thick and hard pericardium, were dissected after making some holes in the pericardium with the burr. Moreover, this equipment made dissection of pericardium from epicardium easier than with scissors, and prevented some damage of myocardium.

Comparison of the postoperative chest roentgenogram with the preoperative one showed that the inferior and posterior calcified pericardium was well removed (Fig 2).

Comment

The key points of successful constrictive pericardiectomy are early operation and as complete a pericardiectomy as possible. Constrictive pericardium is usually dissected with scissors. However, very thick and calcified pericardium cannot be dissected only by scissors; furthermore, myocardium is sometimes damaged or perforated by scissors when they are used on a beating heart.

The high-speed burr we used has the following benefits for dissecting constrictive pericardium: (1) easy handling, (2) light weight, (3) small size, and (4) easy destruction of only the hard tissue such as calcified pericardium. These benefits make it easier to destroy the thick and hard pericardium even in a small operative field such as the posterior or lateral portions of the heart. Additionally, with this burr it is easier to distinguish the epicardium from the pericardium than with scissors, and it did not cause damage to the myocardium because we could feel easily the difference between the hard pericardium and the soft epicardium and myocardium. Some drips of saline solution would be helpful to the manipulation of the burr and prevent heating of the burr head against the hard pericardium.

In conclusion, the small high-speed burr was useful in pericardiectomy of constrictive pericarditis.

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New Technique for the Arterial Switch Operation in Difficult Situations

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The technique described here, a modification of the Aubert operation, avoids coronary reimplantation and also eliminates the need to use artificial material to transfer coronary circulation.

(Ann Thorac Surg 1990;50:1000–1)

Coronary reimplantation is the most important part of the arterial switch operation for patients with transposition of the great arteries, and may contribute substantially to surgical outcome. In the case of single coronary artery and intramural coronary arteries, reimplantation can be particularly difficult.

In 1977, Aubert and associates [1] reported the first successful case using the internal tunnel repair technique, eliminating coronary reimplantation from the arterial switch operation. Recently Kawada and co-workers [2] modified this technique though only for those patients who have difficulties with coronary reimplantation. Here, we propose another simple modification of this coronary rerouting procedure. The use of the viable aortic wall for partitioning facilitates the rerouting procedure without distortion, and a high patency rate and future growth can be expected [3]. With this technique, pulmonary root reconstruction is easier than in the case of coronary transfer, and compression of the coronary arteries can be avoided.

Figure 1 shows this technique in detail. When transecting both great arteries at the level of pulmonary bifurcation (Fig 1a), a flap of the anterior aortic wall on the nonfacing sinus is joined to the distal ascending aorta like a tongue. The posterior aortic wall of the proximal ascending aorta is cut in a U-shape just above the coronary orifice, and adjacent pulmonary arterial wall is also cut in the same fashion. These two U-shaped cuts are anastomosed to form an aortopulmonary window (Fig 1b). A few millimeters along the proximal aorta and the proximal pulmonary artery are sutured on either side of the U-shaped window. After the French maneuver has been done, the flap of distal ascending aorta is sutured to cover the coronary orifice and the surgically created aortopulmonary window so as to form an internal tunnel for coronary circulation (Fig 1c). Then the distal aorta and the proximal pulmonary artery stump are anastomosed. The defect of the anterior proximal aortic wall is repaired by a patch of xenograft pericardium (Fig 1d). Finally, reconstruction of the neopulmonary root is completed.

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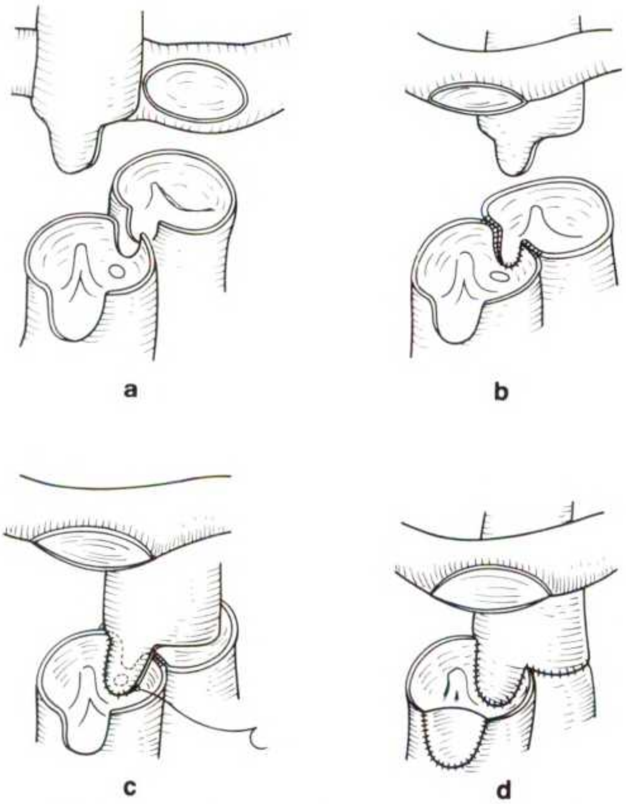

Fig 1. Modification of Aubert operation: (a) transection of both great arteries; (b) creation of aortopulmonary window; (c) construction of coronary rerouting; (d) new aortic root and coronary pathway are completed.

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INVITED COMMENTARY

The publication by Aubert and associates in 1977 described an operation they had successfully performed for transposition of the great arteries. The initial step of this procedure, which was done on cardiopulmonary bypass while the infant was being cooled, consisted of creating a fistula between the aorta and the pulmonary artery. Then the great arteries were transected under circulatory arrest distal to the fistula and a V-shaped patch was used to form the roof of a tunnel from the fistula to the coronary ostia. Finally, the arterial switch was carried out.

Aubert's operation occupies an important place in the history of transposition surgery but, of course, it has been displaced from routine performance by the more satisfying arterial switch with coronary ostial transplantation to the new aorta. The importance of this brief communication by Takeuchi and Katogi is to remind us that the Aubert concept can be used when the coronary arteries cannot be translocated as, for example, with a single coronary ostium and intramural coronary branches running medially and laterally, prohibiting complete coronary dissection and flipping of the coronary button without occluding the coronary branches.

This unusual variant can be dealt with in the manner described in this report if the surgeon examines the coronary origins carefully before transecting the aorta. The coronary ostium in such unusual circumstances can be left intact and perfused via the fistula covered by the aortic flap.

If the great arteries have already been transected before the difficult coronary problem is recognized, then the principles described in an excellent paper by Planché and associates [1] can be applied.

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Transatrial Approach Revisited

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A surgical approach is described to provide excellent exposure of the interior of the left atrium and the mitral valve apparatus.

(Ann Thorac Surg 1990;50:1002–3)

Surgical exposure of the left atrial structures including the mitral valve can be problematic, especially when the left atrium is small. We describe an incision that is a modification of an approach by Dubost and colleagues [1, 2] and provides an excellent exposure of the interior of the left atrium and the mitral valve apparatus.

Technique

Standard cardiopulmonary bypass techniques with bicaval cannulation are used. An incision is begun on the anterior surface of the right superior pulmonary vein approximately 1 cm from the interatrial groove. The initial opening is usually made with a No. 11 blade immediately after cross-clamping the aorta to decompress the left side of the heart while administering cardioplegia solution. After securing tapes or clamps around both cavae, the incision is extended onto the interatrial groove and then obliquely across the free wall of the right atrium (Fig 1). Small retractors are placed within the right atrium to expose the atrial septum. The fossa ovalis is identified, and the septum is incised from the right superior pulmonary vein to the anterior limbus of the fossa ovalis. Care must be taken not to carry the incision above the superior limbus or beyond the anterior limbus of the fossa ovalis (Fig 2). By placing retractors beneath the atrial septum, excellent exposure of the mitral valve and the contents of the left atrium is obtained (Fig 3).

At the completion of the procedure, the septum is closed with a running suture of 4-0 Prolene. Because the septum is often markedly hypertrophied, care must be taken to incorporate the endocardium of both sides of the septum into the suture line. One or two interrupted pledgeted Prolene stitches may be required near the inferior portion of the fossa ovalis if the tissue is especially thin or is torn. This can usually be avoided if proper care is taken in making the initial incision.

This suture is continued to the junction of the septum and the free right atrial wall. Here it is brought to the outside of the right atrium and tied. The right superior pulmonary vein is closed with a separate 4-0 Prolene suture. This suture line may be left untied and may be used to vent the left atrium while removing air from the heart. The right atriotomy is closed with another 4-0 Prolene suture (Fig 4).

When a segment of the atrial septum has been excised as in resection of atrial myxomas, the septal defect is reconstructed with a patch of pericardium or Gore-Tex. The patch is extended out onto the right superior pulmonary vein, then the right atriotomy can be closed separately in the usual fashion.

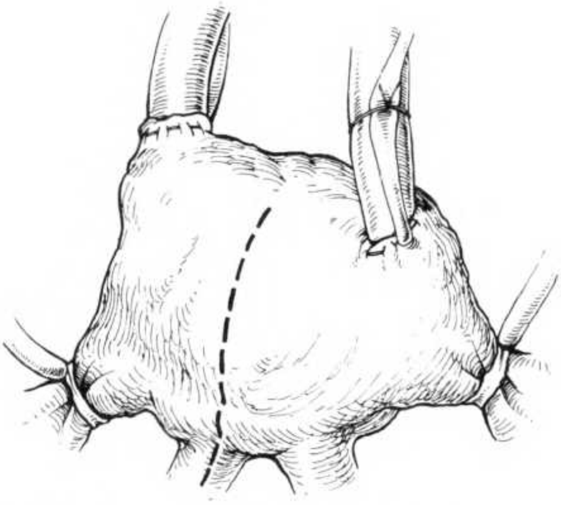

Fig 1. Dotted line depicts the direction of the incision.

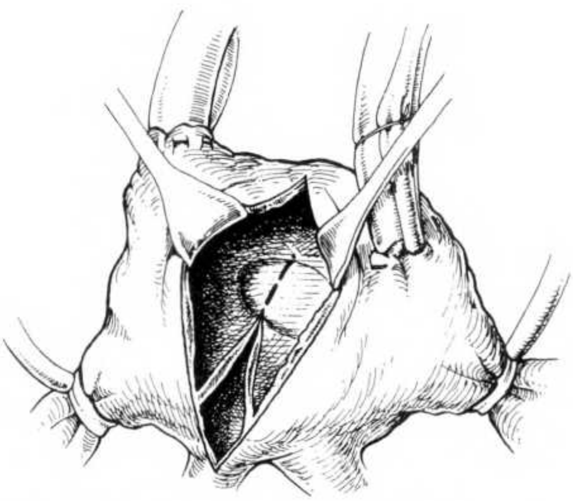

Fig 2. Interatrial septum is incised from the right superior pulmonary vein to the anterior limbus of the fossa ovalis.

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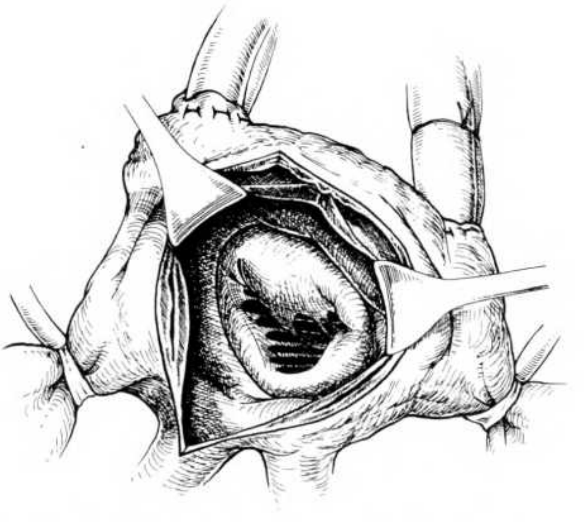

Fig 3. Mitral valve fully exposed without displacing the heart.

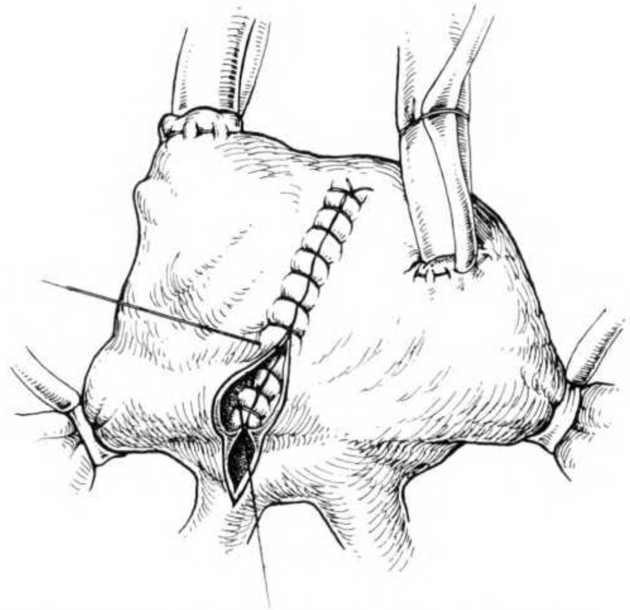

Fig 4. Technique of closure of the incision and venting through the right superior pulmonary vein.

Comment

Over the past 15 years, we have used this transatrial incision in more than 1,000 patients. Originally, this approach was used most often for mitral valve replacement in children or in patients with a very small left atrium. This exposure allows for simultaneous tricuspid valve procedures when indicated, and we have also found this incision to be helpful when resecting atrial myxomas.

In the past several years, we have used this approach exclusively when contemplating mitral valve reparative procedures. The exposure of the valve and subvalvar apparatus is excellent. Because extensive retraction is not required, the valve is not distorted. Therefore a more accurate determination of the degree and location of any residual regurgitation can be made. We have also used the transatrial incision in a recent case involving endocardial resection for Wolff-Parkinson-White syndrome with a left free wall accessory pathway and found it to provide very satisfactory exposure.

This incision appears to be safe. None of our patients demonstrated clinically significant atrial septal defects. No serious arrhythmias or sick sinus syndrome were noted in our patients. The only potential drawback of this approach is the greater length of time required to close the transatrial incision. The excellent exposure provided and the increased ease of performing the primary procedure more than offset this minor inconvenience.

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Operative Insertion of a Transvenous Left Atrial Pressure Monitoring Line

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We describe a technique for the insertion of a left atrial pressure monitoring line during open heart operations in infants. The procedure requires central venous cannulation with a long catheter, followed by intraoperative placement of the catheter tip through the interatrial septum into the left atrium. The technique has been used successfully in 35 infants.

(Ann Thorac Surg 1990;50:1004–5)

The accurate measurement of left and right ventricle preload is of the utmost importance when steering young children through the period of hemodynamic instability that follows an open heart operation. Measurement of left atrial filling pressure has traditionally required the insertion of a catheter through the thoracic wall and through the wall of the right superior pulmonary vein into the left atrium [1]. Such a technique carries a small but serious risk of complications such as hemorrhage.

We present a simple technique for the insertion of a left atrial line that eliminates these problems. It combines the relatively simple procedure of central venous cannulation with intraoperative insertion of the catheter into the left atrium.

Technique

The infant is prepared in the routine manner for open heart operation by the administration of a general anesthetic and administration of a broad-spectrum antibiotic as prophylaxis, followed by central venous cannulation. After this a 30-cm, 20-gauge cannula (Leader Cath, Vygon, Gloucestershire, UK) is then introduced percutaneously according to the Seldinger technique into one of the three major peripheral veins. The femoral vein is the site of choice for cannulation, but when this approach is unsuccessful the internal jugular or subclavian veins are used. The full length of the catheter is advanced into the vein and into the right atrium.

Aseptic technique is practiced during insertion of all central venous catheters, using sterile gloves, 10% povidone iodine to clean the skin, and sterile drapes. After cannulation the site is dressed with a transparent, water-resistant adhesive plastic dressing (Tegaderm, 3M Health Care, Leicestershire, UK; or Niko-Gard, Nikomed, Hampshire, UK).

The second stage of catheter insertion is performed intraoperatively through a right atriotomy. The catheter is trimmed to a length sufficient to cross the interatrial septum into the left atrium without passing through the mitral valve. The tip is then passed through either an existing atrial septal defect, which may be repaired around the catheter, or a small puncture made in an intact septum (Fig 1).

After the operation the left atrial catheter is coupled to a transducer for pressure monitoring; it is labeled identifying its point of termination and forbidding the infusion of drugs through it. In the smallest infants patency is maintained by hourly bolus injection of 1 mL of 5% dextrose solution containing 1 unit of heparin, and in larger infants a continuous infusion is used delivering 3 mL an hour. When no longer required, usually after 2 days, the catheter is removed.

Comment

The transthoracic insertion of a left atrial line may be complicated by hemorrhage and catheter retention, which may at times necessitate an additional thoracotomy to rectify them [1]. To avoid these problems Dr A. D. Crew, consultant anesthetist, and Mr D. R. Walker, consultant cardiothoracic surgeon, developed the technique described. After we had instituted it in our practice, we became aware that McNicholas and Niguidula [2] had previously reported the procedure in this journal in 1983. We are, however, unaware of the widespread practice of the technique, and have therefore provided this report to promote it.

The transvenous left atrial line has been inserted in 35 infants, weighing between 3 to 10 kg, who underwent a corrective open heart operation likely to be complicated by left heart failure. This included congenital abnormalities such as atrioventricular canal defects and transposition of the great arteries. During all such procedures a right atriotomy is required, thus allowing access to the interatrial septum.

Central venous cannulation in babies may prove difficult; we have, however, successfully cannulated the femoral veins of more than half the neonates, and in many more of those older than 3 months. An internal jugular vein was accessed in most of the remainder, with the procedure being abandoned in few infants. The procedure is not time consuming, adding between 5 and 15

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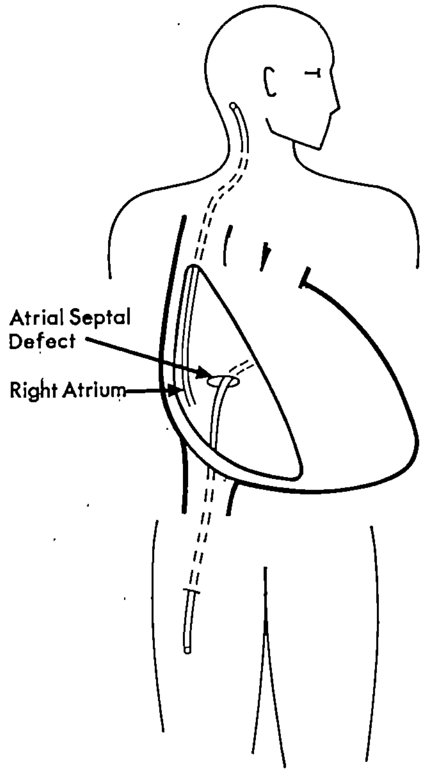

Fig 1. The right atrium is catheterized from one of the three major peripheral veins, the femoral vein being most common. The catheter is directed across the interatrial septum.

minutes to the time required by the anesthetist to prepare the infant for operation.

We have found the use of the transvenous left atrial pressure monitoring catheter, preferentially inserted through the femoral vein, to be relatively free of complications. We have not observed any traumatic or infectious complications. Contamination arising from the perineum is reduced after open heart operations as these patients rarely defecate while critically unwell, and most have a urinary catheter in situ thus minimizing contamination by urine. However, in the event of soiling, meticulous nursing care in addition to protection afforded by the plastic dressing appear to minimize contamination.

Femoral venous cannulation has been reported to be safe and easily performed [3]. Stenzel and associates [4] reported that the incidences of infectious and noninfectious complications were similar after femoral and nonfemoral cannulation. Femoral catheters became infected in 3.7% of the patients and nonfemoral catheters, in 7.3%. None of the infections complicating femoral lines were caused by enteric organisms.

In conclusion, we have found this small but innovative modification of the use of central venous catheters to be of great benefit to our management of infants after open heart operations. The absence of intrathoracic complications leads us to recommend this technique to cardiac surgeons and anesthetists for their regular use.

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Postinfarction Ventricular Septal Defect Repair: Retrospective Thoughts and Historical Perspectives

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Evolution of surgical techniques for repair of postinfarction ventricular septal rupture initially involved differentiation of these lesions from prior experience with surgical approaches to congenital ventricular septal defects, which were in the main not applicable. Second, understanding of the differing anatomical locations of postinfarction ventricular septal defects required innovation in terms of the location of the cardiotomy and type of repair necessary to achieve a successful result in any given patient. The gradual appreciation of different clinical courses pursued by patients after postinfarction ventricular septal rupture both in terms of location of the defect and the degree of right ventricular functional impairment has led to increased urgency relative to the timing of surgical repair. The incorporation of specific anatomical concepts of surgical repair and better understanding of the time course of physiological deterioration of patients can ultimately lead to an integrated approach aimed toward improved salvage of patients suffering this catastrophic complication of acute myocardial infarction.

(Ann Thorac Surg 1990;50:1006–9)

RESECTION OF ACUTE VENTRICULAR ANEURYSM AND RUPTURED INTERVENTRICULAR SEPTUM AFTER MYOCARDIAL INFARCTION*

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LAWRENCE R. BURWELL, M.D.,
DEXTER W. LAWSON, M.D.,
AND W. GERALD AUSTEN, M.D.

After the initial report by Cooley and associates in 1957 [1] of surgical closure of a postinfarction interventricular septal defect, a number of cardiac surgeons [2–7] developed interest in and made contributions to the evolving surgical treatment of postinfarction septal rupture. Most of these early operations were conducted in patients several weeks to months after septal rupture, in individuals who had completely recovered from their infarction, and by selection would fall into a low-risk group as we currently define it. Also, from early experiences with closure of congenital ventricular septal defects, surgeons carrying out these initial operations for postinfarction ventricular septal rupture usually did so through a right ventriculotomy, often with great difficulty because of the resulting limited exposure. The first patient in our hospital's series of postinfarction septal defects was operated on in August 1968 through the same approach and the operation was not successful for similar reasons of difficult exposure. In our institution, Dr W. Gerald Austen performed successful closure of a postinfarction ventricular septal defect in February 1969 in a patient 8 months after infarction, the defect being closed with a patch through an anterior right ventriculotomy. This patient did well for many years thereafter. Another patient was operated on acutely in March 1969, again through an anterior right ventriculotomy, but did not survive because of incomplete closure of the defect.

Discussions among the members of our cardiac surgical group regarding this lesion centered around the critical condition of patients with septal rupture when we would encounter them and the difficulties in technical exposure to the defect through the traditional anterior right ventriculotomy. At about this time there began to appear in the literature a few reports of early or emergency closure of septal perforations [8–11]. Notable among these was Heimbecker and associates' [8] superb early study of infarctectomy and clinical applications to patients with postinfarction ventricular septal defects.

I had joined the cardiac surgical staff at the Massachusetts General Hospital in July 1968 after lengthy clinical training interspersed with substantial research training in the physiology laboratories of Dr Stanley Sarnoff at the National Heart Institute and Dr Theodore Cooper at St. Louis University. Whereas my clinical training had provided a great deal of experience in traditional protocols for approaching clinical problems, my research training under Dr Sarnoff and Dr Cooper had taught me to question standard approaches to clinical problems when those traditional approaches consistently yielded less than satisfactory results. This encouragement by Dr Sarnoff and Dr Cooper to free-associate when confronted with a problem that did not easily yield to conventional protocols, combined with my clinical training, came to a head in September 1969 when I was asked to see a patient 69 years of age who had been admitted to the hospital 10 days earlier with acute anteroapical myocardial infarction. Thereafter the patient had slipped into refractory conges-

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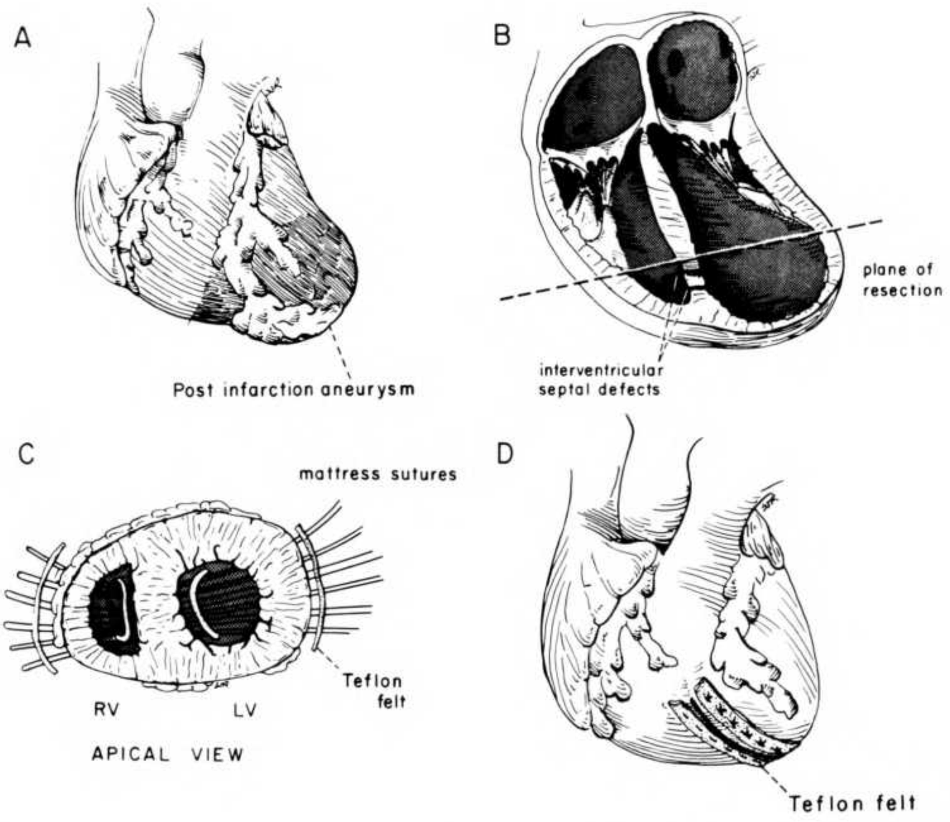

Fig 1. Anterior surface of the heart (A and B), showing the infarct and area of resection, and apical view of the heart (C and D), showing the approximation of right ventricular wall, septum, and left ventricle with mattress sutures buttressed by Teflon felt. (Reprinted by permission of the New England Journal of Medicine *[1970;283:1507–8].)*

tive heart failure in the presence of a documented left to right shunt at the ventricular level. In the next 24 hours progressive hypotension and respiratory failure requiring endotracheal intubation developed. Despite inotropic support, hypotension persisted and the patient became anuric. With the diagnosis of septal rupture established and in the presence of acute hemodynamic deterioration, there seemed little choice but to attempt emergency surgical repair of the lesion in hopes of restoring adequate pump function. I consulted with Dr Austen and Dr Mortimer Buckley, and both encouraged me to proceed and to see what I could accomplish.

At operation on the evening of Sunday, September 4, anesthesia was skillfully induced by Dr John H. L. Bland but was associated with further hypotension necessitating the institution of partial bypass through the femoral vessels in the groin for hemodynamic support. Sternotomy was then performed and complete cardiopulmonary bypass established through a second right atrial drainage line and left ventricular decompression by the right superior pulmonary vein. When the heart was elevated out of the pericardial cavity, the thin-walled necrotic infarct of the apex of the heart could be easily visualized. Blood could actually be seen swirling beneath the thin membrane comprising the center of the infarct. It seemed senseless to perform a right ventriculotomy when the direct approach to the septal defect could be accomplished directly through the infarct, which in any event appeared in danger of imminent rupture. The borders of the infarct, now 12 days old, were well demarcated. The infarct was easily excised, the distal shaggy and necrotic septum debrided along with the two septal perforations, and closure of the heart accomplished as described in our report [12] (Fig 1). The patient's subsequent clinical course was complicated but she ultimately made a complete recovery and lived another 8 years enjoying an active life with her large family.

At about this time, development of the intraaortic balloon pump was undertaken by Avco Corporation and the Massachusetts General Hospital Cardiac Surgical Group. It was arranged by Dr Austen, Dr Buckley, and Dr Arthur Kantrowitz of the Avco-Everett Research Laboratory for the Massachusetts General Hospital to be the principal institution for clinical evaluation of the Avco intraaortic balloon pump. This cardiac support system was implemented here at the Massachusetts General Hospital largely through the efforts of Drs Buckley and Austen [13] and our cardiological colleagues Drs Robert Leinbach and Herman Gold [14, 15]. The initial success of intraaortic balloon pumping to provide support for patients in cardiogenic shock after acute myocardial infarction brought to this institution large numbers of patients referred for mechanical circulatory support in the presence of hemodynamic deterioration after acute myocardial infarction. Among these patients with ventricular power failure were substantial numbers of patients with acute mechanical complications of myocardial infarction including ventricular septal rupture, acute mitral regurgitation, acute ventricular aneurysms, and pure left ventricular power failure. The latter problem was approached successfully by Drs Eldred Mundth and Buckley through a combination of perioperative intraaortic balloon pump support and emergency coronary revascularization with successful results wherein pharmacological support had been previously futile [16]. Drs Buckley and Austen also developed strategies for the successful early treatment of acute ischemic mitral regurgitation [17, 18].

My own interest gravitated toward the patients included in the catch who were found to have ventricular septal rupture. In the subsequent months and few years, we were privileged to treat an inordinately large number of patients with this catastrophic complication of acute myocardial infarction, initially with mixed results but gradually with increasing success as we analyzed our failures and realized that ventricular septal rupture was not a single lesion but rather several, anatomically and in terms of the degree of physiological impairment encountered in different patients. Our successes encouraged us and our failures stimulated us again to free-associate and hopefully therefore to turn a lemon into lemonade.

Although we had achieved a modicum of success by employing the transinfarction left-sided approach [19] to anterior and apical ventricular septal defects with primary closure, we failed to achieve a successful result in our first six attempts to close inferoposterior defects. It was in April 1974 that I returned to the operating room with a patient in deep cardiogenic shock shown to have a posterior septal rupture. Shumacker [20] had earlier suggested a posterior approach to this lesion. After excising the large posterior left ventricular infarct, it seemed obvious that there would be no way to close the resulting defect in the posterior left ventricular free wall (the septal defect itself was easily closed with mattress sutures buttressed with strips of Teflon felt and tied down on the epicardial surface of the diaphragmatic right ventricle, also as suggested by Shumacker) without a degree of tension on the suture line, which had caused rupture and uncontrollable bleeding in our earlier attempts. We were aware of the earlier experimental report of Collins and Collins [21], who in the dog had replaced segments of left ventricular free wall with prosthetic material, and I decided on the spot, in view of our previous failures with primary suture technique, to replace the defect with a circular patch cut from a Cooley tightly woven Dacron aortic tube graft. With the hole in the ventricular wall thus closed, the previously observed ruptured papillary muscle was corrected by mitral valve replacement through a transatrial approach. To our great satisfaction and not a little surprise, the patient was easily separated from cardiopulmonary bypass on continuing intraaortic balloon pump support. This initial success [22] was corroborated by our subsequent experiences, which have been reported elsewhere [23].

It is difficult to know how to ascribe the small stepwise successes interspersed with failures that ultimately over time lead to integrated approaches [24–27] to a difficult clinical problem. From a personal perspective, I believe that this process results from sound clinical training, fundamentally augmented by a different type of training, namely that of problem solving, which is integral to basic investigation. Thus in this particular instance preparation met opportunity in an institution whose traditions fostered academic freedom and open inquiry, wherein the departmental chairman and division director provided a supportive environment and encouraged a young surgeon to go for broke.

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REVIEW OF RECENT BOOKS

Thoracic Trauma

Edited by R. Maurice Hood, Arthur D. Boyd, and Alfred T. Culliford
Philadelphia, W.B. Saunders, 1989
480 pp, illustrated, $95.00

Reviewed by Kamal A. Mansour, MD

This is a comprehensive text authored by members of the faculty of New York University Medical Center. It includes all aspects of thoracic trauma that are managed by cardiothoracic surgeons. It is also a good book for emergency medical technicians, paramedics, house officers with limited experience in trauma surgery, and general surgeons working in the emergency room.

It discusses monitoring and emergency therapeutic measures in acute trauma situations. It stresses early diagnosis and management. It details the radiologic findings in various thoracic trauma patients with proper radiologic correlation to the changing clinical picture. It discusses chest wall injuries from rib fracture to traumatic asphyxia, including the management of hemothorax and pneumothorax from its simplest form to tension pneumothorax and sucking wounds of the chest. Lung injuries are nicely presented with emphasis on the mechanics of injuries caused by high-velocity missiles and blast injuries. However, there is no reference to traumatic atrioventricular fistulas. Penetrating and nonpenetrating cardiac trauma, its protean manifestations, diagnostic considerations, and therapeutic implications are discussed in too much detail with lengthy descriptions. The chapters on traumatic aortic rupture, tracheobronchial injuries, and diaphragmatic and esophageal injuries are too detailed, repetitive, and somewhat opinionated. There is no reference to acute mediastinal shift resulting in serious hemodynamic changes that occur in diaphragmatic herniations with intestinal obstruction. The chapters on pathophysiology of trauma and trauma-associated acute respiratory failure are excellent. Pulmonary and pleural complications, iatrogenic injuries, and anesthetic management, as well as postoperative care, are covered extensively.

However, the text contains a large number of loose, sloppy, and inaccurate proofreading errors. Some errors are very bad and others are not so dangerous but could lead to the wrong information. To state "that the thoracic duct passes through the esophageal hiatus of the diaphragm into the thoracic cavity" is totally wrong as the thoracic duct normally passes through the aortic hiatus and not through the esophageal hiatus. To name Dr Boerhaave's patient, the grand admiral of the Dutch navy "Van Wasserman" instead of "Van Wassenaer" conveys the wrong information. To name the chief minister of King Djoser, the architect of the step pyramid at Saqqara, "Inhotep" instead of "Imhotep" is wrong. To spell out "Lebsche" sternal knife as "Lelesche" is bad. The illustration showing the technique of hilar clamping is incorrect as it shows the inferior pulmonary vein lying between the pulmonary artery and the superior pulmonary vein. It could be an oversight, but to show the same illustration again reflects carelessness. The topics of tension pneumothorax, diaphragmatic injuries, and chylothorax are repeated twice and even three times.

On a serious note, the book is comprehensive, informative, and contains a lot of practical applications. My advice to the senior editor with "gray hair of experience and skill," as he describes himself, is to be more compulsive with his editorial obligations, to cut down redundancy, and to delete repetitions. Taking these changes into consideration, the second edition of this book should belong to the library of all practicing cardiothoracic surgeons and particularly those of us who are involved in emergency surgical practice. And by the way, my name is "Mansour" and not "Marssauir" as quoted!

Atlanta, Georgia

Intraoperative Echocardiography

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Intraoperative echocardiography has emerged as a new technique by which the cardiac surgeon can more precisely define the operative anatomy and physiology, immediately evaluate the results, and more accurately monitor left ventricular function in the operating room. This review summarizes the current applications of intraoperative two-dimensional and Doppler echocardiography in both the epicardial and transesophageal forms, and discusses the advantages and possible limitations of these methods in the practice of clinical cardiac surgery.

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The increasing availability and use of cardiac ultrasound technology have made cardiac surgeons aware that intraoperative echocardiography can provide valuable information during all types of procedures [1]. The use of epicardial or transesophageal (TEE) echocardiography permits real-time visualization of ventricular function, chamber volume, valvular anatomy, and flow dynamics. Echocardiography can be employed at the beginning of the operation to confirm or better define anatomical abnormalities. During the operation, it provides the surgeon with instant feedback, which can result in important modifications in surgical procedures. Finally, echocardiography allows the surgeon to monitor global and regional ventricular function so that postoperative hemodynamic therapy can be based on a more accurate assessment of the patient's immediate clinical state. This review summarizes the current applications of intraoperative echocardiography and discusses its advantages and limitations in the practice of clinical cardiac surgery.

History of Intraoperative Echocardiography

In 1972, Johnson and co-workers [2] first described the use of intraoperative echocardiography; they used M-mode echocardiography to evaluate the results of mitral valve operations. Spotnitz and associates [3, 4] used a similar method to measure left ventricular compliance and further define intraoperative anatomy in patients with tetralogy of Fallot. In 1982, Spotnitz [1] advanced the technique when he reported his experience with two-dimensional echocardiography in the assessment of left ventricular function in patients undergoing coronary artery bypass grafting (CABG), the evaluation of prosthetic valve function, and the localization of myxomas and other foreign bodies. This study documented the versatility of intraoperative echocardiography and its potential importance for cardiac surgeons.

Echocardiographic Techniques

Two-Dimensional Echocardiography

Two-dimensional echocardiography allows the surgeon to visualize both the endocardium and the epicardium of the left ventricle without distortion or manipulation of the myocardium. Unlike angiography and nuclear imaging techniques, two-dimensional echocardiography allows visualization of myocardial segments at multiple levels and in several different projections. It can provide assessment of qualitative regional wall motion (RWM) by demonstrating changes in endocardial wall movement or systolic wall thickening. Quantitative changes in segmental ventricular systolic function can be calculated from computer programs that analyze changes in radial wall thickening or endocardial motion over a single cardiac cycle. Global function can be assessed by analyzing changes in cross-sectional areas between diastole and systole (ejection fraction index) as well as by combining long-axis sections with short-axis sections to derive ventricular volumes to calculate ejection fractions based on geometric modeling.

Doppler Echocardiography

Doppler echocardiography provides information about the flow of blood within the various heart chambers, across the valves, and into the great vessels. The Doppler effect, first described by Christian Johann Doppler in 1842, is based on the principle that transmitted sound will reflect from a moving object and return to the point of transmission at a frequency shift relative to the velocity and direction of the moving object. In the case of cardiac Doppler analysis, red blood cells act as the moving objects that reflect the ultrasound emitted by the echo transducer. Cells moving toward the transducer will compress the ultrasonic wave fronts and the reflected sound will return to the transducer at a frequency shifted upward. Conversely, a lower returning frequency indicates that the blood cells are moving away from the transducer. The higher the blood velocity, the greater the frequency shift. The difference in frequency is known as the Doppler shift.

A further application of the Doppler technique is its ability to determine pressure gradients across narrow orifices based on the modified Bernoulli equation. This

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formula is based on the fact that blood velocities across narrow orifices are directly proportional to the pressure gradients across these orifices [5] and is described as $P = 4V^2$, where P is the pressure gradient across the orifice in millimeters of mercury and V is the velocity of blood distal to the stenosis in meters per second. This equation allows for the rapid and accurate estimation of pressure gradients across stenotic and regurgitant valves as well as intracardiac shunts, and forms the basis for much of the hemodynamic information derived from Doppler studies.

Pulsed Doppler echocardiography measures low-velocity flow, which can be useful in evaluating cardiac output across various orifices. It can be performed in a sequential fashion or in real time to evaluate large chamber areas. In 1982, Namekama and colleagues [6] developed a real-time color-flow mapping system allowing Doppler detection of intracardiac blood flow to be superimposed on a two-dimensional echocardiographic display. There are various conventions for displaying blood velocities. The most popular type at present portrays blood moving toward the transducer as various shades of red or orange and blood moving away from the transducer as blue. The lighter the shade of color, the higher the velocity.

VALVULAR STENOSIS. The Doppler technique is best used to assess valvular stenosis and regurgitation. When a stenotic valve is present, the antegrade velocity across the valve is increased. The peak gradient across the stenotic valve can be obtained from the magnitude of the increased velocity. Continuous-wave Doppler study can measure peak and mean pressure gradients and, in combination with two-dimensional imaging, can determine valve areas with a high degree of accuracy.

VALVULAR REGURGITATION. Valvular insufficiency can be assessed by determining the area of regurgitant flow in the upstream chamber or the height or width of the jet using either pulsed or color Doppler techniques [7–10]. Because regurgitant jets are often eccentric in their projection, it is important to examine the valve from as many angles as possible. In the case of mitral regurgitation, Helmcke and colleagues [7] demonstrated a close correlation of transesophageal color Doppler echocardiography with cardiac catheterization using orthogonal imaging planes. Measurement of regurgitant areas appears to be the most predictive color Doppler function for assessing the severity of mitral regurgitation [7–9]. Aortic insufficiency is assessed by evaluating regurgitant jet height or area in the distal left ventricular outflow tract [10].

In addition to evaluating valve function, transesophageal color Doppler echocardiography has also been applied to the visualization of coronary artery blood flow. In a recent study [11], clear color images of the left main trunk were obtained in 32 of 36 patients. This technique may allow clinicians to visualize abnormal flow through stenotic coronary arteries, as well as to assess the patency of bypass grafts. At present, transesophageal Doppler echocardiography usually provides visualization of only the left main trunk, proximal-middle circumflex, proximal left anterior descending, and right coronary arteries. Evaluation of more distal coronary artery anatomy will require the implementation of higher resolution transducers. The new high-frequency annular array probes may facilitate this application.

Epicardial Echocardiography

Intraoperative echocardiograms can be obtained using either the epicardial or the transesophageal method. In the former technique, the echo transducer is either gas sterilized or placed in a sterile plastic sleeve. In the latter case, ultrasonic gel is placed between the transducer surface and the inside of the sleeve to improve the acoustic interface between the outer surface of the sleeve and the epicardium. In our experience and that of others [12], this method has resulted in no infections or serious arrhythmias requiring cardioversion. In general, we prefer to have the patient cannulated while epicardial echocardiograms are obtained in case transducer-induced arrhythmias should occur.

Several standard echocardiographic positions have been established to allow the surgeon to see most anatomical landmarks. The parasternal view along the anterior surface of the right ventricle provides a tomographic view of the anterior and posterior mitral valve leaflets, mitral annulus, and chordae tendineae. This is the preferred view to assess mitral valve prolapse and a flail leaflet. If the transducer is rotated 90 degrees, a short-axis or cross-sectional view is obtained. In this manner, "breadloaf-sliced" cuts of the mitral valve and left ventricle can be obtained. The mitral valve view is useful for assessing orifice area and leaflet redundancy. The mid-papillary short-axis view includes segments of the myocardium supplied by all three coronary arteries and is the best single view with which to monitor RWM abnormalities associated with ischemia [13]. The aortopulmonary sulcus view is probably the best angle for the detection of a mitral regurgitation "jet." The aorta–superior vena cava view is especially useful for visualizing aortic and left ventricular outflow pathology. The right ventricular four-chamber view, obtained by placing the transducer on the inferior wall of the right ventricle, can accurately depict tricuspid valve pathology.

Transesophageal Echocardiography

Over the past decade, TEE has developed into an important intraoperative echocardiographic technique and is currently supplanting epicardial hand-held techniques as the method of choice for intraoperative echocardiographic assessment. The incorporation of a miniature echo transducer into a flexible gastroscope allows an ultrasound beam to be directed in multiple planes from any position within the esophagus (Figs 1, 2). The proximity of the esophageal lumen to most cardiac structures provides an unimpeded ultrasonic pathway to the heart in contrast to the often limited windows available using the standard transthoracic technique. The pioneering work of Schluter and co-workers [14] demonstrated that TEE is capable of producing high-resolution two-dimensional images. In addition, the recent incorporation of color Doppler echo-

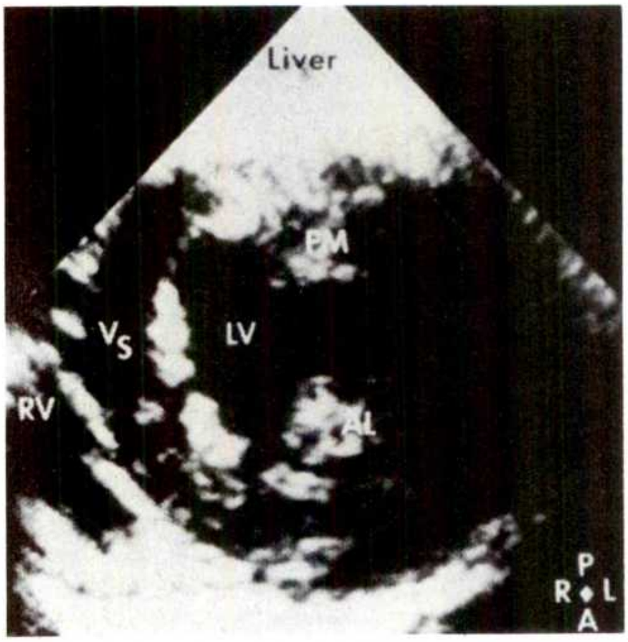

Fig 1. Transesophageal echocardiography: cross-sectional view. (AL = *anterior-lateral muscle;* LV = *left ventricle;* PM = *papillary muscle;* RV = *right ventricle;* VS = *ventricular septum.*)

cardiography has increased the power of the technique by an order of magnitude.

Transesophageal echocardiography has several advantages over the epicardial method for intraoperative studies. Once the probe is positioned after intubation, the surgeon need not interrupt the operation to obtain an echocardiographic study. The probe does not threaten sterility of the operative field, and transesophageal echocardiographic imaging can continue throughout the entire procedure. By keeping the esophageal probe in the same position throughout the procedure, several studies at the same anatomical position can be obtained. This constant positioning is critical if sequential evaluation of segmental wall motion is desired to detect the development of myocardial ischemia. Another advantage of TEE is its lack of arrhythmogenicity versus the epicardial approach. Finally, the probe can be left in place and used to monitor postoperative myocardial function in the intensive care unit.

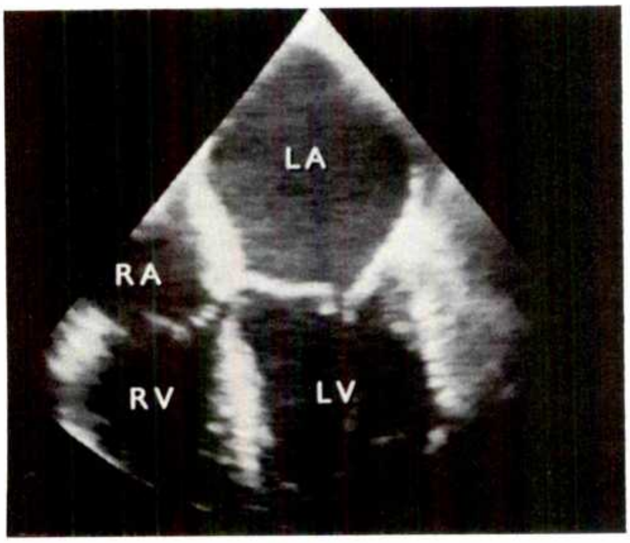

Fig 2. Transesophageal echocardiography: four-chamber view. (LA = *left atrium;* LV = *left ventricle;* RA = *right atrium;* RV = *right ventricle.*)

There are certain limitations to the transesophageal technique. First, the anatomical relationship between the esophagus and the heart restricts the number of imaging planes available. This problem may be alleviated with the development of biplanar transesophageal echocardiographic probes. Second, because an adequate long-axis image of the left ventricle from mitral annulus to apex is rarely obtainable, volumetric analysis based on the usual geometric modeling (bullet formula or Simpson's method) is not possible, and it is necessary to rely on short-axis cross-sectional measurements to obtain a global ejection fraction index [15], which provides a somewhat limited estimate of systolic function. Third, TEE has been limited to children older than 8 years because of the size of the probe. However, smaller probes incorporating fewer transducer elements are now being clinically tested [12] and have recently been released into commercial production. Fourth, transesophageal probes are not as accurate as their transthoracic counterparts in visualizing the basal ventricular segments or the right ventricular outflow tract and the distal descending aorta. Despite these limitations, the ease of insertion and the minimal interference with surgical techniques make the transesophageal approach generally more practical than the epicardial approach for intraoperative monitoring.

Contrast Echocardiography

Contrast echocardiography involves the injection of microbubbles of air into a cardiac chamber to define the blood pool and, therefore, chamber anatomy. These echogenic "microcavitations" can be followed to assess cardiac flow and the presence of intracardiac shunts. Originally, investigators used agitated saline solution, blood, or indocyanine green as contrast agents. In these cases, the microcavitation size was relatively large and the contrast medium was absorbed in the pulmonary capillary bed, thereby preventing the imaging of the left-sided structures. Keller and co-workers [16] have developed sonication techniques to prepare echo contrast agents capable of capillary transit with resultant left-sided passage. Sonication is performed using a commercially available sonicator, which creates cavitation waves and, subsequently, bubbles within the liquid. These cavitations instantly collapse, and a second generation of bubbles is created. To date, injection of echogenic contrast agents into the right ventricle has been done safely in humans [17].

Goldman and Mindich [18] were the first to report the use of intraoperative myocardial contrast echocardiography during CABG procedures. Because a sonicator was not used, the size of the air bubbles was variable.

Recent advances in sonicator design allow the size and quantity of the microbubbles to be controlled. The microbubbles produced by this newer technique are similar in size to red blood cells and therefore behave as true intravascular tracers of red blood cell flow. Using this new technique in the canine myocardium, Kaul and coworkers [19] were able to show that the measurement of the transit time of microbubbles through the myocardium with two-dimensional echocardiography accurately reflects regional myocardial blood flow.

In an experimental study, the same research group [20] used Renografin-76 (diatrizoate meglumine and diatrizoate sodium) in a canine model to demonstrate that intraoperative myocardial contrast echocardiography could effectively delineate flow through the native coronary arteries and internal mammary artery grafts in both a qualitative (visual) and quantitative (computer-derived analysis) manner. Smith and co-workers [21] injected sonicated Renografin-76 into vein grafts so that graft function and patency could be observed after CABG. In addition, the myocardial area perfused by each vein graft was visualized. Matthew and colleagues [22] demonstrated that contrast echocardiography could also be used to assess the quality of cardioplegia distribution during CABG.

Intraoperative contrast echocardiography is still an investigational technique, but it may have an important role during CABG procedures. It may be helpful in localizing intramyocardial coronary vessels, in localizing epicardial vessels in extremely fatty hearts, and during reoperations in which coronary vessels are poorly visualized as a result of extensive pericarditis. In vessels that are diffusely diseased or where tandem lesions are present, it may facilitate the location of areas that are relatively free from disease and thus help to plan the site of the anastomosis. By imaging the anastomotic site, the surgeon can identify and correct technical errors before the patient leaves the operating room. Finally, the adequacy of cardioplegia distribution can be ascertained and the most critical areas grafted first to ensure more homogeneous distribution of cardioplegia and to achieve better myocardial protection.

Uses of Intraoperative Echocardiography

Assessment of Valve Function

Intraoperative echocardiography has probably been of greatest value in the assessment of valve repair. The heightened interest in valve repair, primarily in the mitral position, requires that surgeons be able to accurately assess the adequacy of the procedure in the operating room. Previously employed evaluative methods were often crude, providing only gross assessment of valve integrity and poor predictability of clinical outcome. Injecting fluid into the arrested ventricle to check for valvular leakage into an upstream chamber was inaccurate because of altered, nonphysiological chamber geometry and pressure in the nonbeating heart. Measurement of left atrial pressure and height of V waves after discontinuation of cardiopulmonary bypass was often misleading, as these variables are dependent on atrial size and compliance as well as on ventricular preload and afterload.

Two-dimensional echocardiography with Doppler color flow can be of tremendous help to the surgeon for both valve repair and replacement in the operating room. Before the institution of bypass, echocardiography can determine the precise anatomy of the mitral leaflets, depicting leaflet redundancy, prolapse, and flail condition, as well as chordal and papillary muscle integrity. In addition, the degree or presence of annular dilatation can be assessed. This information can help to determine whether valve repair or replacement would be more suitable and, if a repair is to be performed, the specific technique that should be employed. After cardiopulmonary bypass, echocardiography, in combination with color Doppler imaging, can assess the adequacy of either the repair or the replacement. If persistent regurgitation is observed, echocardiography can often document the cause and guide further intervention.

Before the advent of color Doppler imaging, injection of contrast medium was the major method used to evaluate mitral valve repair. In an early study using contrast echocardiography, Mindich and co-workers [23] demonstrated that this technique could detect small degrees of mitral regurgitation when conventional methods failed to do so and allowed more precise repair of intraoperatively defined residual mitral regurgitation. To obtain detectable bubbles, 5 mL of agitated 5% dextrose in water was injected into the left ventricle through a transseptal needle. In the absence of mitral regurgitation, virtually all of the microbubbles exited through the aorta. In the presence of regurgitation, a mass of microbubbles was seen to reflux into the left atrium.

The introduction of color Doppler echocardiography offered several advantages over contrast echocardiography in the assessment of mitral regurgitation. This new technique did not necessitate the insertion of intracardiac needles or the injection of contrast agents. In addition, the echocardiographic analysis could be performed without interruption of the operation. Beat by beat analysis was now possible, and long-term follow-up studies could be performed using similar methods of evaluation. In a series of 56 patients, Mauer and colleagues [24] examined the ability of color Doppler echocardiography to assess the severity of mitral regurgitation at the time of operation and to evaluate the adequacy of mitral valve repair before closure of the chest. They compared the Doppler technique with a method that involved filling the arrested ventricle with saline solution to check for mitral valve integrity. The fluid-filling technique failed to detect any degree of intraoperative mitral regurgitation; however, a significant number of patients thought to have had an adequate repair were found to have severe mitral regurgitation in the postoperative period. Intraoperative color Doppler echocardiography, however, demonstrated a significant correlation with postoperative studies.

It is important that in any intraoperative assessment of mitral regurgitation, care be taken to match the preload and afterload conditions of the initial prebypass study, as the degree of mitral regurgitation is highly dependent on arterial systolic pressure and ventricular volume. Volume repletion and administration of vasopressors may be necessary to obtain loading conditions comparable with those found in daily life. Only when this is performed can assessment be carried out in a physiologically reliable manner.

To assess the utility of TEE during cardiac valve operations, Sheikh and co-workers [25] at Duke University studied 154 patients undergoing cardiac valve procedures. In this series, the prebypass intraoperative echocardiograms revealed unsuspected findings that led to some modification of the planned operation in 12% of the patients. In 7% of the patients, an inadequate valve repair was identified before the chest was closed, and this finding resulted in reinstitution of bypass and an additional reconstruction or replacement. In total, surgical decisions were made in 27% of the patients based solely on findings from intraoperative TEE. The greatest impact was seen in mitral valve reconstructions. Sheikh and co-workers concluded that intraoperative TEE with color-flow Doppler echocardiography was extremely useful in planning surgical procedures, assessing results, and identifying patients who may be at risk for postoperative complications.

Doppler echocardiography also provides a powerful tool in the assessment of prosthetic valve function. Epicardial probes can easily visualize aortic prostheses, but mitral valve regurgitation can be masked by strong echo "shadowing" caused by reflections of the prosthesis itself. With the recent introduction of TEE, there is better visualization of mitral regurgitation jets in the left atrium, thereby dramatically enhancing the evaluation of prosthetic mitral valve dysfunction [26].

Intraoperative echocardiography can also help to determine ventricular hemodynamics after valve repair or replacement [27, 28]. In addition, it can help to determine the long-term effects of early periprosthetic leaks. Recent work from our laboratory [29] indicates that periprosthetic regurgitant leaks are commonly found by TEE after valve replacement. Do these leaks persist, do they deteriorate further, and do they prognosticate future valve dehiscence? Does Doppler echocardiographic evaluation of regurgitation after valve repair in the immediate post-pump period accurately predict regurgitant degree in a later more hemodynamically stable setting where ventricular filling is adequate and afterload increased? No studies comparing intraoperative color Doppler with postoperative color Doppler echocardiography and ventriculography have yet been reported. Clearly, further investigative studies of normal surgical results are required to elucidate the clinical implications of these intraoperative echocardiographic findings.

Assessment of Myocardial Ischemia: Segmental Wall Motion and Global Function

Two-dimensional echocardiography is a sensitive and accurate detector of changes in regional and global ventricular function. Van Daele and co-workers [30] studied 98 patients during anesthesia induction before CABG. They simultaneously monitored pulmonary capillary wedge pressure, a standard 12-lead electrocardiogram, and left ventricular wall motion using TEE. In their study, TEE was the most sensitive indicator of myocardial ischemia, followed by the electrocardiogram and, finally, pulmonary capillary wedge pressure. Rea and associates [31] also used intraoperative echocardiography to detect wall motion changes in patients undergoing CABG. They found a significant decrease in intraoperative ejection fraction and new segmental wall motion abnormalities in patients with postoperative electrocardiographic and enzyme changes, and concluded that intraoperative two-dimensional echocardiography is a safe and accurate method to evaluate the immediate effects of CABG on left ventricular function, to detect postoperative abnormalities, and to guide in postoperative care.

We [32] recently employed intraoperative two-dimensional echocardiography in 17 patients undergoing CABG to study the effects of coronary artery revascularization on segmental wall motion. We used a qualitative visual scoring system in the evaluation of endocardial RWM as well as digital-computer analysis of systolic segmental thickening percentage and global systolic function. We found that in those segments with ischemic dysfunction before CABG, there was a significant increase in quantitative RWM after CABG. These results are similar to those of intraoperative studies by Topol [33], Koolen [34], and their associates, which showed that immediately after surgical revascularization, there is a significant improvement in systolic thickening in segments with preoperative ischemic dysfunction. Such analysis of segmental wall motion may provide important information in the immediate postbypass period. If new areas of ischemic dysfunction are found after bypass, the surgeon should be alerted to investigate the grafts to make sure there are no technical problems before closure of the chest. Also, Doppler detection of increased postoperative mitral regurgitation may indicate papillary muscle dysfunction, suggesting the need for further revascularization of the posterior myocardium or a mitral valve procedure.

Our intraoperative findings were in agreement with earlier studies [35, 36] showing that qualitative visual RWM interpretation lacks the sensitivity and precision associated with changes in systolic thickening in evaluating ischemic endocardial motion. The computerized analysis of RWM required in the evaluation of segmental thickening is probably a much more accurate way to assess myocardial ischemia than gross visual estimation. However, currently the hardware and software available for on-line analysis of RWM are limited. Development of dedicated echocardiographic digitization and analysis equipment has begun and will eventually allow for on-line beat to beat quantitative calculations of RWM. Built-in video memory will allow echocardiographic images to be displayed at baseline and with side-by-side sequential updates. As a result, it will be possible to compare RWM before and after CABG both qualitatively and quantitatively. Such analysis will also be useful to the anesthesiologist who is monitoring high-risk cardiac patients during noncardiac operations.

In addition to evaluating the results of CABG procedures, we have found intraoperative echocardiography useful in the immediate postbypass period where hypotension is often a problem. Intraoperative echocardiography can help to determine whether this is due to peripheral vasodilatation or alterations in myocardial contractility. The surgeon can determine whether volume replacement is necessary or whether inotropic agents should be instituted. If there is global ventricular dysfunction seen on echocardiograms early in the postbypass period, we have chosen earlier insertion of the intraaortic balloon pump and other mechanical support systems rather than increments in inotropic agents. Finally, the echo probe can be left in position for monitoring purposes in the intensive care unit to help the surgeon determine when doses of inotropic agents can be tapered and when the patient can be weaned from mechanical devices.

Assessment of Congenital Heart Disease

The combination of two-dimensional echocardiography and color Doppler imaging is uniquely suited for evaluating the quality of surgical repair in congenital heart disease, as these techniques provide real-time, high-resolution images of complex anatomical anomalies, intracardiac shunts, and indications of gradient severity across stenotic areas. In the prebypass period, echocardiography can be used to provide information regarding the anatomy and flow characteristics of septal defects, atrioventricular canals, tetralogy of Fallot, truncus arteriosus, great vessel transposition, single ventricle, and pulmonary, aortic, and mitral valve abnormalities [37]. After bypass, it can detect residual shunts, regurgitant valvular lesions, and periprosthetic leaks.

To determine the usefulness of intraoperative echocardiography in surgical decision making during congenital procedures, Ungerleider and associates [38] studied 224 patients undergoing repair of congenital defects using epicardial color-flow Doppler echocardiography. Echocardiographic results were judged helpful when they contributed information that either "settled preoperative uncertainty or provided data that influenced the conduct of the operation." In this series, intraoperative echocardiographic studies detected 52 new and unsuspected findings in 47 patients (21%) compared with preoperative catheterization or transthoracic echocardiographic data. Echocardiographic studies had some effect on the operative procedure in 105 (47%) of the 224 patients. In 38 patients (17%), postbypass echocardiographic studies demonstrated residual defects, and additional repairs were attempted in 30 patients. Follow-up studies showed that patients who left the operating room with a major residual defect noted on postbypass echocardiographic studies had a significantly increased incidence of reoperation or death. Patients who left the operating room with normal echocardiographic findings had a 90% likelihood of a long-term acceptable outcome compared with a 65% long-term acceptable outcome in patients with abnormal echocardiographic findings at the conclusion of the procedure. In a recent study from the Mayo Clinic [39], color Doppler echocardiography and two-dimensional echocardiography were also found to be useful for assessing surgical results, especially in determining residual shunts and atrioventricular valve regurgitation.

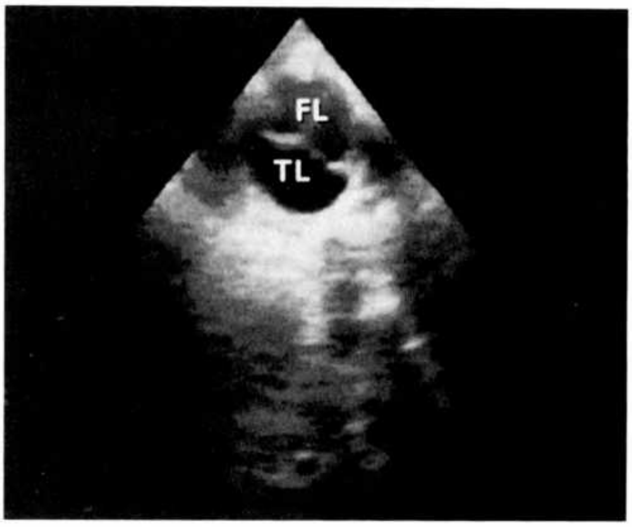

Fig 3. Dissecting ascending aortic aneurysm. (FL = *false lumen;* TL = *true lumen.)*

Despite the initial enthusiasm for the use of echocardiography in the repair of congenital defects, there are some limitations in the existing techniques. Dense surgical prosthetic patches can frequently limit echocardiographic and Doppler windows. Transesophageal studies have been limited in the pediatric age group because of the size of the existing probes, although Kyo and colleagues [40] recently used TEE in 8 patients ranging in weight from 3.9 to 27 kg without any complications. Future modifications in transesophageal technology will make this an even more viable tool for surgeons to implement during congenital heart operations.

Management of Aortic Dissection

Two-dimensional echocardiography has been a useful diagnostic tool in the intraoperative management of dissecting aortic aneurysms. Aortic angiography is time-consuming and requires the use of contrast agents and transportation of critically ill patients to the catheterization laboratory. Computed tomographic scans also require contrast medium and do not always visualize the intimal tear. Visceral branches are not seen, and the degree of aortic insufficiency cannot be assessed. Transesophageal two-dimensional echocardiography offers several advantages over angiography and computed tomographic scanning. Echocardiography can provide an accurate diagnosis of an aortic dissection within 15 minutes of a patient's admission to the emergency room [41]. The site of the intimal tear as well as the extent of the dissection can often be localized (Fig 3). In addition, the presence of aortic insufficiency and pericardial effusions can be easily detected.

There are two limitations with the echocardiographic technique. First, the upper ascending aorta and the arch vessels are relative "blind spots," although the complementary addition of suprasternal transthoracic echocardiography may eliminate this problem. Second, if branch vessels are involved, angiography can better determine the surgical anatomy. Nevertheless, in a recent series of 164 patients with suspected aortic dissection, Erbel and co-workers [41] showed that TEE was 99% sensitive and 98% specific in making the diagnosis as opposed to aortography, which had an 88% sensitivity and a 94% specificity.

Takamoto and colleagues [42] have used color-flow Doppler imaging to better visualize the true and false lumens in the dissecting aneurysm. In a series of 11 patients, they used intraoperative color Doppler echocardiography to visualize the site of the dissection and the flow dynamics in the true and the false lumen to help plan the operative approach. Using the same technique in 33 patients, this same group [43] found it especially helpful in localizing the site and extent of dissection as well as determining the degree of aortic valve involvement.

Detection of Complications of Myocardial Infarction

Two-dimensional echocardiography in combination with color-flow mapping has become a major diagnostic tool for the detection of mechanical complications of myocardial infarction including ventricular septal defects (Fig 4), mitral regurgitation (Fig 5), and the presence of mural thrombi, and for the detection of ventricular aneurysm and pseudoaneurysm formation. It provides a rapid diagnostic assessment that can be done at the bedside in the face of deteriorating hemodynamics. Serial studies can be started preoperatively and extended into the intraoperative and postoperative periods. If coronary angiography is necessary, echocardiography can eliminate the need for a left ventricular contrast study.

In the operating room, echocardiography helps to further localize the infarction-associated lesion and provides an immediate assessment of the surgical repair. Color Doppler flow mapping has been especially helpful in facilitating the diagnosis of left ventricular pseudoaneurysms [44]. Transmyocardial blood flow can easily be seen passing across the wall defect. This is particularly important when the rupture is small and cannot be visualized by two-dimensional imaging alone. Doppler echocardiography has also helped to visualize left ventricular thrombi [45] as well as to determine the site and number of acute ventricular septal defects [44].

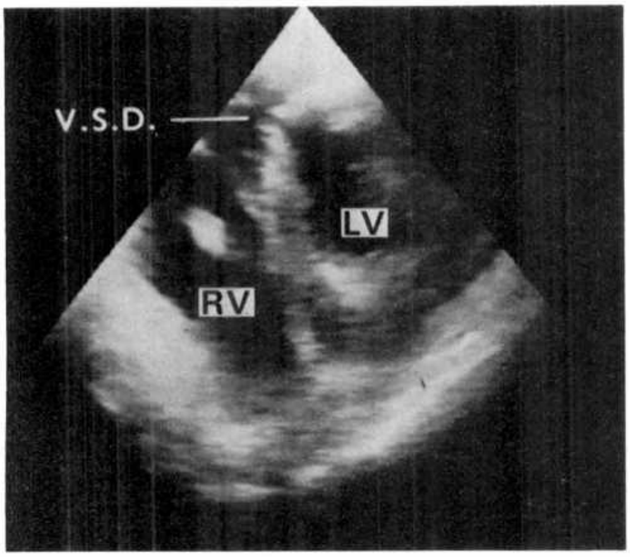

Fig 4. Acute myocardial infarction: ventricular septal defect (V.S.D.). (LV = *left ventricle;* RV = *right ventricle.)*

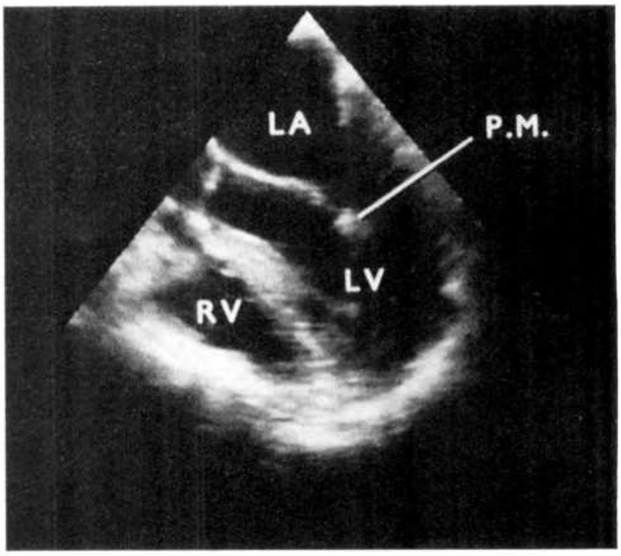

Fig 5. Acute myocardial infarction: ruptured papillary muscle (P.M.). (LA = *left atrium;* LV = *left ventricle;* RV = *right ventricle.)*

Identification of Tumors and Foreign Bodies

Intraoperative two-dimensional echocardiography has been especially helpful in localizing the site and number of cardiac tumors such as myxomas and fibroelastomas [1, 46]. Foreign bodies such as bullets and fragments can be detected more easily, and therefore, the site of the intramyocardial incision can be accurately determined. We recently encountered a patient with a renal cell carcinoma extending into the inferior vena cava and the right atrium. Intraoperative TEE helped to alert us to the fact that tumor had replaced nearly the entire right atrium. We were able to plan our cannulation sites and atrial incisions in such a way that the tumor could be completely excised.

Conclusion

Intraoperative two-dimensional echocardiography and Doppler echocardiography have emerged as important tools for the cardiac surgeon. These techniques help in decision making regarding the type of surgical procedure that will be required, provide an immediate assessment of the accuracy of the procedure, and aid in monitoring left ventricular hemodynamics and determining therapeutic interventions in the postoperative period. Future modifications in transducer design and computerized image analysis will make echocardiography an even more powerful force in shaping intraoperative surgical decision making.

We acknowledge the secretarial support of Deborah Cammarata, who assisted in the preparation of the manuscript.

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REVIEW OF RECENT BOOKS

Surgery of the Thymus

Edited by Marco Merlini, David B. Clarke, and Michael Dusmet
Heidelberg, Springer-Verlag Berlin, 1990
345 pp, illustrated, $175.00

Reviewed by Donald G. Mulder, MD

The thymus gland has been and remains today somewhat of an enigma because we are not certain of all its varied functions in either the normal or the diseased state. Yet much is known about the thymus, and it is the objective of this book to provide a broad-based perspective as we view our understanding of this intriguing endocrine organ.

The chapters are clustered into 8 parts beginning with the embryology, anatomy, and histology as well as the physiology and immunology of the gland. Parts II and III deal with the surgical pathology with a heavy emphasis on thymic tumors and related diseases. Part IV focuses on clinical investigations, primarily radiologic (conventional, computed tomographic scan, magnetic resonance imaging) and neurophysiologic diagnostic techniques. Parts V, VI, and VII are of more interest to surgeons because they discuss the operative management of patients undergoing thymectomy in myasthenia gravis, including those with thymic tumors, as well as some speculation about the effects of thymectomy in human oncogenesis.

This book has something here for the anatomist, pathologist, endocrinologist, neurologist, radiologist, and anesthesiologist as well as the surgeon for whom the text is said to be primarily oriented. From the surgeon's standpoint the thymus is of particular interest as it relates to myasthenia gravis, thymic tumors, and organ transplantation. These areas among others are covered in considerable detail and with fairness in areas of controversy. Although there are 42 contributors to this text of whom the majority are from Europe, each of the 32 chapters is thoroughly referenced with relevant scientific articles from all countries including the United States. As in any multiauthored text there are substantial areas of redundancy and overlap. This is most apparent in chapters 10 (Neuroendocrine, germ cell, and nonepithelial tumors), 11 (Mediastinal tumor-like conditions and tumors that can simulate thymic neoplasms), and 31 (Postoperative evolution and prognosis of thymic tumors). More rigid editing would have minimized this duplication. However this modest flaw is more than compensated for by the overall quality of the text and its practical as well as speculative contributions. Any serious student of the thymus gland, including thoracic surgeons and cardiothoracic residents, will find this a valuable reference book.

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CORRESPONDENCE

Spasm of Arterial Conduits in Aorta–Coronary Bypass Grafting

To the Editor:

Suma [1] and Mills and Everson [2] suggest that the gastroepiploic artery (GEA) is more prone to vasospasm than the internal mammary artery. Recently we [3] have conducted comparative anatomical studies among various arterial conduits in aorta–coronary bypass grafting. Some of our findings seem to support the clinical observations as reported by them.

In contrast to the almost strictly elastic appearance of the media of the internal mammary artery, we have observed the abundant presence of smooth muscle cells in the media of the right GEA and the inferior epigastric artery, both of which are used as alternative arterial conduits in myocardial revascularization [1, 2, 4, 5]. Based on these findings we hypothesize that the GEA and inferior epigastric artery, which have an important vasoregulatory function in the perfusion of the stomach and the abdominal wall, respectively, are more prone to vasoconstriction than the internal mammary artery. In contrast, the latter vessel, being in the vicinity of the aorta, serves more as a pressure reservoir by means of the elastic properties of its wall.

In our experience, spasm of the internal mammary artery is not as vigorous as that observed in the GEA by Suma and by Mills and Everson. In reference to the observation of the latter two authors that the free GEA graft is more prone to vasospasm than the in situ GEA graft, we hypothesize that vasospasm in the free GEA may represent an autoregulatory mechanism of reduction of arterial flow as a reaction to exposure to the high central aortic pressure and powerful pulsatile hemodynamics, in contrast to the lower arterial pressure and damped hemodynamics in the GEA in its natural environment.

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Reply

To the Editor:

Spasm of the coronary artery bypass graft is an important problem, as is coronary artery spasm. Figure 1 demonstrates a possible spasm that occurred in the free right gastroepiploic artery (GEA) graft in my current experience.

To know the reason why GEA is prone to spasm, histological study could be an important approach. As Mills and Everson [1] and I [2] described, elastic fibers are rare in the media of the GEA, whereas they are rich in the internal thoracic artery. In their recent article, van Son and colleagues [3] reported that smooth muscle cells are rich in GEA but are rare in internal thoracic artery. These histological differences might be one of the factors responsible for the spasmogenicity of GEA.

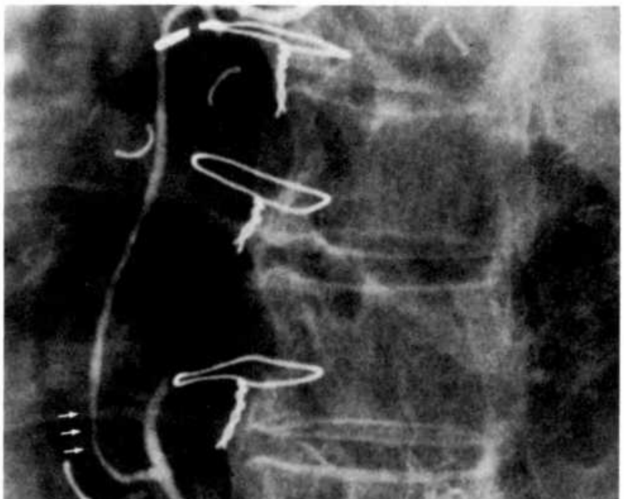

*Fig 1. Free right GEA graft was anastomosed to the main right coronary artery in combination with the left internal thoracic artery graft to the anterior descending coronary artery in a 72-year-old woman who had severe varicose veins. Diameter of GEA was more than 2.5 mm at the time of operation, and the GEA was not forcefully dilated at its preparation. At 3 weeks after operation, the free GEA graft was patent but possible spasm was found at the distal part of the graft (*arrows*). Nitroglycerin injection was not attempted in this case.*

On the other hand, study of pharmacological response in autologous grafts is an important counterpart to solve the problem. Though my colleagues and I [4] reported that the contractile responses of GEA to noradrenaline, ergonovine, and serotonin were similar to those of internal thoracic artery, we recently found different response between these two arteries to histamine. Internal thoracic artery was constricted by histamine, but GEA was not. With increased use of autologous "live" arterial conduits in coronary artery bypass grafting, histological and pharmacological investigation to identify the physiological and anatomical characteristics of each vessel is quite important because graft spasm can be prevented by knowing spasmogenic characteristics of various conduits and maximal graft flow can be obtained with the most powerful chemical dilator for each artery. I believe van Son and colleagues made an important step in this field.

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Ann Thorac Surg 1990;50:1019–23 • 0003-4975/90/$3.50

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Chest Wall Recurrence of Lung Cancer After Transthoracic Fine Needle Aspiration Biopsy

To the Editor:

In a recent article, Seyfer and associates [1] described a patient who had chest wall implantation of lung cancer after fine-needle aspiration biopsy. I would like to document another example of this complication with the following case history of a patient from our practice.

An asymptomatic 65-year-old woman underwent annual physical examination, which was entirely normal. Chest roentgenogram (Fig 1) revealed a 2-cm cavitary lesion in the posterior segment of the right upper lobe. On computed tomographic scan no other pulmonary lesions were seen and there was no hilar or mediastinal lymphadenopathy. She had smoked cigarettes for over 40 years. Fiberoptic bronchoscopy was normal. She underwent fine-needle aspiration biopsy of the lung lesion through the right posterior chest wall with three passes of a 22-gauge spinal needle. Because pneumothorax resulted, a chest tube was inserted, the lung was reexpanded, and she was admitted to the hospital. Cytological evaluation of the aspirate was reported as "adenocarcinoma." Bone, brain, and liver scans were normal. Pulmonary function studies indicated that she would tolerate right upper lobectomy.

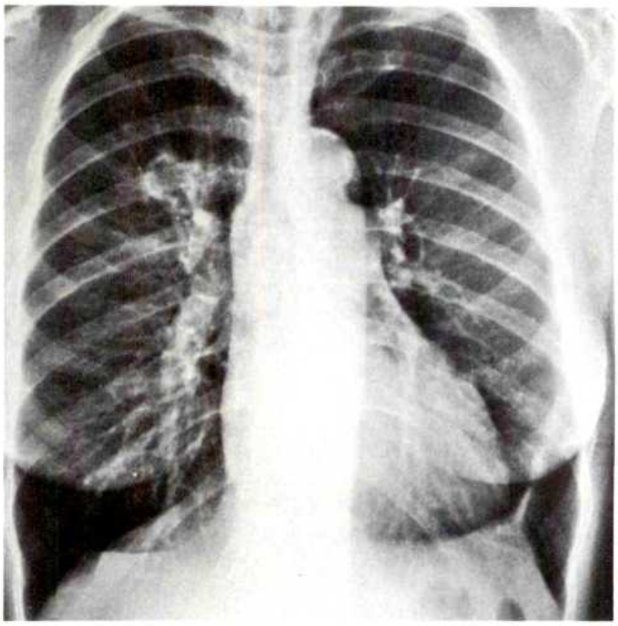
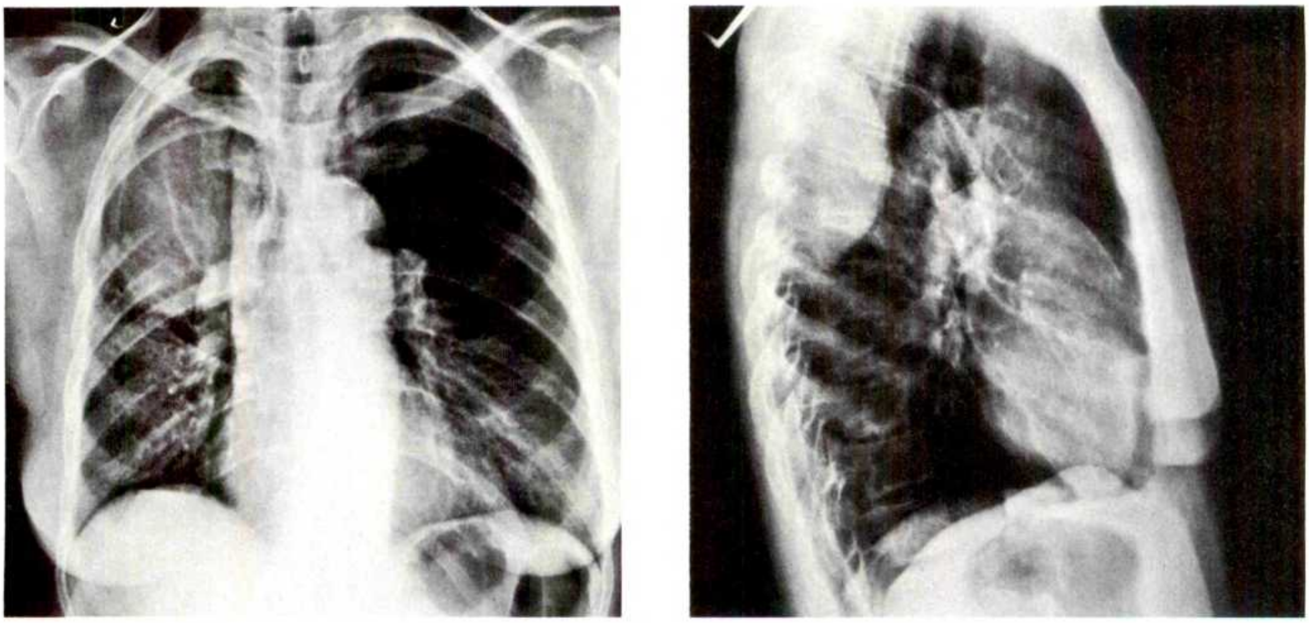


Fig 1. Cavitary lung cancer in the posterior segment of the right upper lobe.

One week later she underwent right thoracotomy. When the pleural space was entered the lung collapsed and no pleural adhesions were found. A 2-cm mass was palpated in the posterior segment of the right upper lobe. The visceral pleura was not grossly involved. No other pulmonary lesions were felt. There was no visible or palpable hilar or mediastinal lymphadenopathy. Right upper lobectomy was carried out. Gross pathological



A

B

Fig 2. Posteroanterior (A) and lateral (B) chest roentgenograms showing chest wall recurrence with rib destruction.

description of the resected tissue was of a "subpleural 2.5 cm diameter tumor with a central 1 cm diameter cavity . . . no extension through the pleura is seen." Microscopic examination revealed poorly differentiated squamous cell carcinoma with no metastases in four hilar lymph nodes. The pleural, bronchial, and vascular margins were free of tumor. No lymphatic or vascular invasion was present. The resected tumor was staged as T1 N0 M0, stage I. No further treatment was recommended.

Seven months later she was seen in the clinic complaining of weight loss, dyspnea, and pain in the right posterior chest wall. A tender 4 × 7-cm mass was palpated posteriorly in the right upper chest wall. Chest roentgenogram revealed a large right posterior chest wall mass, with destruction of the posterior portions of ribs 5 through 7 (Fig 2). This mass was at the site of the previous needle aspiration. Aspiration biopsy of the mass revealed non-small cell carcinoma. Metastatic work-up was negative. She received radiation therapy to the chest wall recurrence and died from disseminated disease 6 months later.

In this case the evidence for implantation, as opposed to direct extension, seems conclusive. The aspiration was performed at the site of recurrence. A pneumothorax was produced, indicating that the lung was not adherent to the chest wall. At operation the lung was completely free from the chest wall and the tumor did not grossly extend to the visceral pleura. This was confirmed by histological examination. The tumor was not invaded nor disrupted during the lobectomy.

Although rare, the potential for tumor implantation must be seriously considered when fine-needle aspiration is recommended for diagnosis of lung lesions with a high likelihood of being malignant, especially when the patient is operable and the lesion is resectable for a potential cure [2].

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Plastic Struts for Delayed Sternal Closure

To the Editor:

Fanning and associates [1] reported that delayed sternal closure may be lifesaving in the management of patients with cardiac dilatation after open heart operations. In less severe cases, simply leaving the sternum unsutured and closing the skin provided sufficient room for the enlarged heart to function satisfactorily. More severe degrees of cardiac dilatation or the presence of stiff, engorged lungs required more space, and in these cases the wound was left open because closure of the skin tended to pull the sternal edges together. The wound was covered with a plastic drape or prosthetic material between the skin edges. Such cases can be difficult to manage because the sternum is unstable and movement may cause cardiac compression, and also because the open wound requires special attention.

Recently 2 of my patients (one adult, one child) who were on maximal inotropic and vasodilator support were not able to tolerate sternal closure at the end of a cardiac operation because of cardiac dilatation and lung engorgement. Closure of the skin alone, even after mobilization, was also not tolerated. Therefore the sternal edges were splinted open using the following technique: a length of stiff intercostal drainage tube about 5 cm (2 inches) long was cut (Meredith No. 22 for the pediatric patient and No. 28 for the adult patient) and a stainless steel wire was threaded through it. The ends of the wire were inserted parasternally on each side from the inside of the chest, brought out, and twisted together presternally (Fig 1). The sternal edges were thus immobilized at a fixed distance. Two such wired struts were used for the child and three for the adult patient. The skin that was mobilized could now be closed without compromising the cardiac output. Both patients were returned to the cardiac intensive care ward where inotrope doses were reduced. Some days later the struts could be removed and the sternal wounds closed.

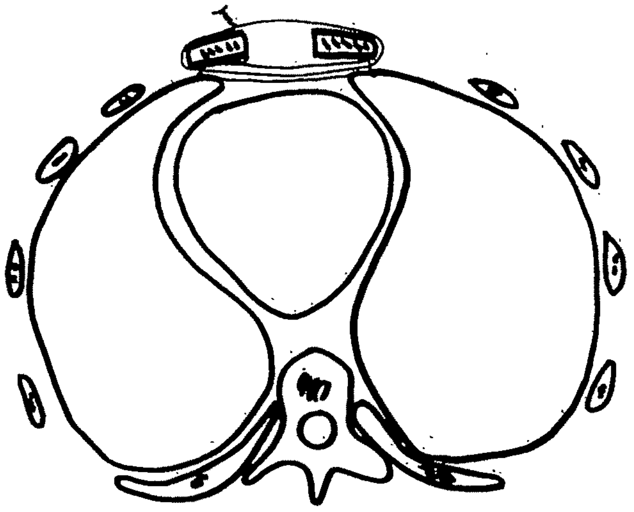

Fig 1. Transverse section through the chest to show the strut and the wire keeping the sternal edges separated by a fixed distance.

With this technique management of these patients was facilitated, particularly the physiotherapy, transport, and nursing of these patients because (1) the sternum was remarkably stable and (2) skin closure was achieved.

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How Should the Risk of Bank Blood Transfusion in Open Heart Operations Be Minimized?

To the Editor:

We read with interest the work of Alajmo and associates [1] concerning blood salvage in cardiac operations obtained by means of infusion of aprotinin (Trasylol) in association with autotransfusion. Their results are confirmed by our experience, here briefly summarized especially with regard to the clinical requisites for autotransfusion, the application of this method, and the remarkable reduction in the incidence of posttransfusion hepatitis.

From September 1986 to July 1990, 500 patients undergoing elective open heart operations were admitted into this study. Blood was collected from 400 of these (group A) and deposited, and the remaining 100 (group B) were also given aprotinin. There were 366 male patients, and the average age was 56 years. Patients with the following clinical or hematologic data were excluded: hemoglobin level less than 120 g/L (12 g/dL) or hematocrit less than 0.35; noncompensated diabetes; severe liver disease; systolic pressure greater than 170 mm Hg or diastolic pressure greater than 100 mm Hg; unstable angina; New York Heart Association functional class IV or ejection fraction less than 0.40; left main coronary artery markedly narrowed (>75%); and transaortic gradient greater than 100 mm Hg.

Three hundred fifty milliliters of blood was collected from the patients who had given their informed written consent at intervals of 3 to 4 days in the hospital. The units collected were separated into red blood cells and plasma, which was stored frozen. Thirty ischemic patients who had a negative history of arrhythmias were subjected to dynamic electrocardiogram before, during, and after each blood donation to evaluate the onset and type of cardiac arrhythmias.

All patients were given orally 525 mg of ferrous sulfate twice daily, from the start of donation up to 10 days after the operation. All patients were operated on within 15 to 21 days after the first donation. Sixty-three percent of the patients underwent myocardial revascularization; 22.8%, valvar operation; 7.4%, radical correction of congenital cardiopathies; and 6.8%, other associated procedures.

To avoid transfusion with banked blood as far as possible, preoperative normovolemic hemodilution was also carried out, whenever possible, as well as filtration and retransfusion of the blood drained from the operative field (during and after the operation).

The total dose of aprotinin administered intravenously was 5 million units (2 before, 2 during, and 1 after extracorporeal circulation). Blood loss was reintegrated initially with 1,050 mL of autotransfusion.

In group A the average overall time of extracorporeal circulation was 92 minutes; the mean hematocrit values (basal, preoperative, and at the time of discharge) were 0.42 ± 0.03, 0.34 ± 0.02, and 0.30 ± 0.03. The mean overall blood loss was 870 mL, and 14 patients were reopened as a result of bleeding (in 4 cases the source of loss was not identified); 119 patients also received banked blood or plasma (2.6 units/patient). Non-A, non-B hepatitis was observed in 5 of 119 patients, reactions to transfusion were observed in 7 of 119 patients, and isoalloimmunization was observed in 1 of 119 patients.

In group B the overall time of extracorporeal circulation and the mean hematocrit values were substantially similar to those of group A, but the mean overall blood loss was 415 mL (−52.3%). One patient was reopened as a result of bleeding; 9 patients were transfused with 1.6 units/patient of homologous blood or plasma (for 7 of these the period of surveillance for infection passed without complications, whereas for the other 2 no signs of infection have appeared at 5 months).

It is known that the incidence of posttransfusion hepatitis is directly proportional to the number of units transfused. A prospective study carried out in our center on 744 patients revealed an incidence of non-A, non-B hepatitis of 12.7% within 2 months of the operation. The use of autotransfusion in the 400 patients in group A lowered the incidence of non-A, non-B hepatitis to 4.2%. In the remaining 100 patients (group B) with the addition of aprotinin there was no incidence of hepatitis at 5 months.

We conclude that autotransfusion and aprotinin administration proved to be risk-free and easy to perform and also reduced transfusion requirements in patients undergoing open heart operations, minimizing the risk of hepatitis.

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Reference

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Ventricular Perforation With Valvoplasty

To the Editor:

The article in the May issue of *The Annals of Thoracic Surgery* by Robertson and associates [1] entitled "Fatal left ventricular perforation during mitral balloon valvoplasty" is worthy of comment. We recently had a similar case at George Washington University in a 51-year-old woman. In our circumstance, however, rather than attempt to take a dying patient to the operating room, the Bard CPS system was immediately installed, thus preserving pressure, perfusion, and oxygenation during that critical time between perforation and definitive surgical therapy. In the circumstance where a myocardial infarction is in progress, immediate cardiopulmonary bypass may preserve the brain but not sustain the heart. Our patient arrived in the operating room in a relatively stable state, her ventricular perforation was closed and her mitral valve replaced, and she made a relatively smooth recovery.

It is appropriate to emphasize to our colleagues the message of their report in our reply, that is, that surgical standby for these more risky procedures is a must, and our experience would indicate that rapid induction of cardiopulmonary bypass, even by a femorofemoral route, can make a clear difference in survival.

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Reference

1. Robertson JM, de Virgilio C, French W, Ruiz C, Nelson RJ. Fatal left ventricular perforation during mitral balloon valvoplasty. Ann Thorac Surg 1990;49:819–21.

Reply

To the Editor:

My colleagues and I were not surprised to hear of a similar case to ours, presented by Dr Aaron and Dr Alyono. We have found in our discussions with other institutions that several other cases similar to ours have occurred. Therefore the incidence of this condition may be higher than once thought. However, we were pleased to hear of the very satisfactory outcome of the patient experiencing this complication at the George Washington University Medical Center. We feel that the Bard CPS system as used by the George Washington University group clearly allowed for a very satisfactory surgical outcome. However, we did not have the Bard CPS system at the Harbor/UCLA Medical Center at the time our patient experienced the fatal perforation, but believe that perhaps had the system existed, we may very well have been able to salvage the patient in a manner similar to that of Drs Aaron and Alyono.

We certainly concur with their message to the surgical community that mitral balloon valvoplasty is not an innocuous procedure and definitely requires surgical standby. We believe that in institutions where the Bard CPS system exists this may be a very satisfactory way of treating such a life-threatening catheterization laboratory complication.

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Individual Ligation Technique

To the Editor:

I enjoyed reading Dr Penfield Faber's review of the anatomical studies by Brian Blades and Edward Kent in 1940 to develop individual ligation techniques for lobectomy [1]. "This was an important departure," said Dr Faber, "from the previously described mass ligation techniques." But the priorities and original conceptions were not quite so clear as described.

It is easily verifiable that Hugh Morriston Davies of University College Hospital, London, performed a right lower lobectomy by the individual ligation technique in 1912, 28 years earlier [2, 3]. The patient was a man, aged 44 years, with squamous cell carcinoma of the lung. "The various structures at the pedicle of the lower lobe were ligated separately," Davies described the procedure, "and the lobe containing the growth, together with a portion of the parietal pleura, was removed. The proximal end of the bronchus was stitched over and covered with an adjacent portion of lung. The patient's condition was quite good for the first six days; he then developed an empyema, and died on the eighth day. At the autopsy no evidence of leakage from the bronchus could be obtained" [2]. Unfortunately the importance of draining the pleural space postoperatively was not yet recognized. Mr Davies must certainly have dissected the hilum in cadavers; it is inconceivable that he could have traversed such unknown territory without having mapped his way beforehand. Two more lobectomies done by others using the same technique, both unsuccessful, were reported later.

Successful lobectomy with individual ligation was accomplished by Dr E. D. Churchill of Boston on August 10, 1931, 9 years before the study of Blades and Kent. "The lower and middle lobes of the right lung were removed after isolation and separate ligation of the branches of the pulmonary vessels supplying them. The stump of the bronchus was fixed by two sharp hooks and a continuous suture of chromicized catgut used to close the aperture. The peribronchial and mediastinal tissue was drawn over the line of suture by interrupted mattress sutures" [4].

Pneumonectomy by the individual ligation technique followed soon after. Two different surgeons accomplished it successfully in July 1933, 7 years before Blades and Kent. Technically, Dr Edward Archibald of Montreal was first, having done the operation on July 7, 1933 [5, 6]. I am indebted to Dr John J. White of Loma Linda University for pointing this out to me, after I had listed priorities erroneously in another publication. Dr William F. Rienhoff of Johns Hopkins accomplished the same feat almost simultaneously, on July 24, 1933 [7]. At least two previous attempts, in 1915 and 1922, had ended with death of the patients [6].

For every thing there is a season, said the Preacher, and besides, hardly anything under the sun is new. Almost invariably, a "new" conception or finding turns out to have had other antecedents.

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THE SOCIETY OF THORACIC SURGEONS

Twenty-seventh Annual Meeting
February 18–20, 1991
Moscone Center, San Francisco, California

The Twenty-seventh Annual Meeting of The Society of Thoracic Surgeons will be held at the Moscone Center on February 18–20, 1991, preceded by the Twenty-fourth Postgraduate Program on Sunday, February 17. The meeting is open to all duly qualified physicians. Both members and nonmembers are urged to register in advance.

The 1990 Program Committee is very pleased with the response to the abstract call, counting 306 abstracts submitted. In addition to the quantity, the high quality of the abstracts made possible the development of a program that represents all aspects of adult and pediatric cardiac surgery and general thoracic surgery. The utilization of breakout sessions introduced very successfully at the last Annual Meeting is being expanded. On Sunday afternoon, immediately following the Postgraduate Program, there will be an important session entitled "STS National Database—A Significant Tool for Clinical Practice." On Monday afternoon, there will be parallel sessions entitled "General Thoracic I," "Heart Transplant Research," and "Adult Cardiac I," running simultaneously from 1:30 to 2:45.

On Tuesday afternoon, all sessions will run from 1:30 to 5:00 PM. Session I will include General Thoracic II, followed by Lung Transplant Research. Session II will include Cardiac Research, followed by Electrophysiology and Cardiac Research, and Session III includes Adult Cardiac II, followed by Congenital Cardiac.

The popular Tuesday morning Problem Case Presentations and the Wednesday morning Clinical Workshops will continue. In addition, on both Tuesday and Wednesday there will be small group sessions on CPT Nomenclature and the coding process.

The Committee had a significant increase in the number of video tapes submitted for the Annual Movie Night, making it possible to develop an excellent program representative of all aspects of the specialty. The movie night session will be in the Hilton Hotel.

At 11:15 AM on Tuesday, Harry Schwartz, PhD, columnist for *American Medical News*, will present the first Ralph D. Alley Lecture entitled, "The Future of American Thoracic Surgery."

Registration and Fees

An advance registration form with hotel reservation information and details regarding spouses' activities will be mailed to Society members. Nonmembers should write to the Secretary, Richard P. Anderson, MD, The Society of Thoracic Surgeons, 401 N Michigan Ave, Chicago, IL 60611.

Members are not required to pay a registration fee for the Scientific Program. Residents and fellows may register without payment of a registration fee upon presentation of a letter from their chief of service. The fee for other physicians is $250, except guest speakers, authors, coauthors, and invited discussants. Nurses and paramedical personnel may register upon payment of a $40 fee and presentation of a letter of introduction from a Society member.

The Postgraduate Program is sponsored by the Committee on Postgraduate Education of The Society. Registration fee, which includes lunch, for the Postgraduate Program is $65 for all physicians, residents, and fellows.

There will be no advance registration for the concurrent Problem Case Presentations on Tuesday, February 19, or the Clinical Workshops on Wednesday, February 20. Attendance will be by ticket only, and each seminar is limited to 35 physicians. Tickets can be purchased at the Society's registration desk on Monday, February 18, starting at 12:30 PM. A continental breakfast is included.

Presentation and Publication

Papers presented at the Scientific Session must be submitted to the Secretary at the time of presentation, or may be submitted to the Editor prior to the meeting for possible earlier publication.

Speakers for the Scientific Sessions should limit their presentations to 10 minutes. They are requested to meet with the projectionist in the preview room prior to the opening of their session to prepare their slides for presentation.

Those who wish to discuss a paper may write the Secretary prior to the meeting. Discussants from the floor should submit their names to the Moderator at the podium prior to the opening of the session during which the paper is to be presented. Discussants from the floor are limited to 2 minutes; primary discussants are limited to 3 minutes.

Discussants will receive a copy of their remarks shortly after the Annual Meeting. Any delay in the return of the corrected discussion to the Editor will hold up publication of the paper. Timely response of all authors and discussants is, therefore, urged since delays are unfair to those who promptly return their material for publication.

Accreditation

The Society of Thoracic Surgeons is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians. One credit hour in Category I of the Physician's Recognition Award of the American Medical Association may be claimed for each hour of participation by the individual physician. The Postgraduate Program is authorized for eight hours of Category I CME credit.

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TENTATIVE TWENTY-FOURTH POSTGRADUATE PROGRAM THE SOCIETY OF THORACIC SURGEONS

Sunday, February 17, 1991
Moscone Center, San Francisco, California

8:00–12:00 **Morning Session**

8:00 Introduction to the 1991 Postgraduate Course
Andrew S. Wechsler, Richmond, VA, Co-Chairman, Postgraduate Committee

I. Mini Symposium: Advanced Cardiac Valve Surgery
Moderator: Andrew S. Wechsler, Richmond, VA

8:10 Myocardial Protection for Complex Valve and Valve-Coronary Grafting Procedures
Nicholas T. Kouchoukos, St. Louis, MO

8:30 Allograft Aortic Valve and Aortic Root Replacement
Donald B. Doty, Salt Lake City, UT

8:50 The Densely Calcified Mitral Valve and Annulus
Noel L. Mills, New Orleans, LA

9:10 Aortic Valve Replacement With Major Aortic Valve and Aortic Root Calcification
John B. Flege, Jr, Cincinnati, OH

9:30 Tricuspid Regurgitation: Assessment and Intraoperative Management
Lawrence H. Cohn, Boston, MA

9:50 Repeat Operations for Cardiac Valve Disease—Tricks of the Trade
D. Craig Miller, Stanford, CA

11:00 **II. C.C.C.E.T.S. Basic Science Lecture***
Introduction
George C. Kaiser, St. Louis, MO, Chairman, Coordinating Committee for Continuing Education in Thoracic Surgery

Vascular Regulation by Mediators From the Endothelium
Professor Sir John Vane, FRS,** The William Harvey Research Institute, St. Bartholomew's Hospital Medical School, London, England

12:00–1:30 Luncheon

Luncheon Address: Future Directions in Cardiovascular Health Care
Jack M. Matloff, Los Angeles, CA

1:30–5:00 **Afternoon Session**

Introduction
Kent W. Jones, Salt Lake City, UT, Co-Chairman, Postgraduate Committee

III. Mini Symposium: Treatment of Congenital Pulmonary Disorders
Moderator: Kent W. Jones, Salt Lake City, UT

1:30 Tracheoesophageal Fistula
Darroch W. O. Moores, Albany, NY

1:50 Bronchopulmonary Foregut Malformations
Larry R. Kaiser,** St. Louis, MO

2:10 Congenital Tracheal Stenosis
Robert M. Filler,** Toronto, Ont, Canada

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* Open Session

** By Invitation

2:30 Lobar Emphysema and Cystic Adenomatoid Malformation
J. Alex Haller, Jr,** Baltimore, MD

2:50 Panel Discussion: Questions and Answers

IV. State of the Art Lectures
Moderator: Kent W. Jones, Salt Lake City, UT

3:45 Bronchoplastic Techniques
Jean Deslauriers, Ste-Foy, Que, Canada

4:05 Surgical Treatment of Esophageal Motility Disorders
David J. Sugarbaker,** Boston, MA

4:25 Automatic Internal Converter-Defibrillators
Ralph J. Damiano,** Richmond, VA

4:45 Panel Discussion: Questions and Answers

5:10–6:15 **STS National Database**

A Significant Tool for Clinical Practice
Richard E. Clark, Pittsburgh, PA

TENTATIVE PROGRAM
TWENTY-SEVENTH ANNUAL MEETING
THE SOCIETY OF THORACIC SURGEONS

February 18–20, 1991

February 18, 1991 (Monday)
Morning

8:00 Business Meeting (members only)

8:05 Welcome

8:15–11:15 **Scientific Session**

1. **J. Maxwell Chamberlain Memorial Paper**

   The Role of Staging in Prognosis and Management of Thymoma
   Earle W. Wilkins, Hermes C. Grillo, J. Gordon Scannell, Ashby C. Moncure, and Douglas J. Mathisen, Boston, MA

2. Sequential Bilateral Lung Transplantation: Results From 16 Consecutive Patients
   Larry R. Kaiser, Michael K. Pasque, Carolyn M. Dresler, Elbert P. Trulock, Anastasios Triantafillou, and Joel D. Cooper, St. Louis, MO

3. Staged Repair of Diffuse Aneurysm of Aorta Using "Elephant Trunk" Principle for Ascending and Arch Replacement
   E. Stanley Crawford, Lars G. Svensson, Joseph S. Coselli, Kenneth R. Hess, Hazim J. Safi, and Frederick A. Meadors, Houston, TX

4. Inferior Epigastric Artery as a Coronary Conduit
   Hendrick B. Barner, Keith S. Naunheim, Andrew C. Fiore, and Howard H. Harris, St. Louis, MO

5. Surgery of Fulminant Pulmonary Embolism: Indication, Diagnostics, Technique, and Results
   C. Schmid, S. Zietlo, T. O. F. Wagner, J. Laas, and H. G. Borst, Hannover, Germany

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** By Invitation

6. Results of Fontan Operation for Univentricular Heart
Amram Cohen, John Dyck, Dolores Poppe, Jeffrey Smallhorn, Robert Freedom, George Trusler, John Coles, Fred Moes, Ivan Rebeyka, and William Williams, Washington, DC

7. Indications and Limits of Aortic Valve Reconstruction
Carlos G. Duran, Naresh Kumar, Begonia Gometza, and Zohair Al Halees, Riyadh, Saudi Arabia

11:15–12 Noon **Presidential Address**
W. Gerald Rainer

Afternoon

1:30–5:00 **Parallel Scientific Sessions**
Session I

1:30–2:45 **General Thoracic I**
Moderator: Douglas J. Mathisen, Boston, MA

8. Main Bronchial Sleeve Resection With Pulmonary Conservation
Joseph R. Newton, Jr, Hermes C. Grillo, and Douglas J. Mathisen, Boston, MA

9. TNM Staging and Long-Term Follow-up After Sleeve Resection for Bronchogenic Carcinoma
P. Van Schil, A. Brutel de la Riviere, P. Knaepen, H. van Swieten, J. Defauw, and J. van den Bosch, Nieuwegein, Netherlands

10. Bronchopleural Fistula After Stapled Closure of the Bronchus
S. Russell Vester, L. Penfield Faber, C. Frederick Kittle, William H. Warren, and Robert J. Jensik, Chicago, IL

11. Multiple Primary Lung Carcinomas: Treatment and Prognosis
Todd K. Rosengart, Nael Martini, Pierre Ghosn, and Michael E. Burt, New York, NY

12. CUSA (Cavitron Ultrasonic Suctioning Aspirator) for Lung Resection
Gary T. Verazin, Anne-Marie Regal, Joseph G. Antkowiak, Zafar Parvez, and Hiroshi Takita, Buffalo, NY

Session II

1:30–2:45 **Heart Transplant Research**
Moderator: R. Morton Bolman III, Minneapolis, MN

13. Noninvasive Detection of Experimental Heart Transplant Rejection With Positron Emission Tomography
Steven J. Hoff, James R. Stewart, William H. Frist, Robert Kessler, Martin Sandler, James B. Atkinson, John A. Carey, and Walter H. Merrill, Nashville, TN

14. Weight Is Not an Accurate Criterion for Adult Cardiac Transplant Size Matching
Barry B. K. Chan, Kirk J. Fleischer, V. Colt Peyton, Terry L. Flanagan, Curtis G. Tribble, James Bergin, Robert S. Gibson, and Irving L. Kron, Charlottesville, VA

15. UW Solution for Human Donor Heart Preservation: Initial Clinical Experience
Valluvan Jeevanandam, Mark L. Barr, Gary Y. Ott, Juan A. Sanchez, Charles C. Marboe, Joseph S. Auteri, Craig R. Smith, and Eric A. Rose, New York, NY

16. Triiodothyronine Enhances Left Ventricular Performance in Injured, but Not Normal Hearts
Cornelius M. Dyke, Thomas Yeh, Jr, Jon Lehman, Rebecca Dignan, Mai Ding, Anwar S. Abd-Elfattah, Andrew S. Wechsler, and David Salter, Richmond, VA

17. First Successful Bridge to Cardiac Transplantation Using Direct Mechanical Ventricular Actuation
James E. Lowe, Mark P. Anstadt, Peter Van Tright, Peter K. Smith, Paul J. Hendry, Mark D. Plunkett, and George L. Anstadt, Durham, NC

Session III

1:30–2:45 **Adult Cardiac I**
Moderator: Alexander S. Geha, Cleveland, OH

18. The Right Gastroepiploic Artery as a Coronary Artery Bypass Graft
John Pym, John O. Parker, and Roxroy O. West, Kingston, Ont, Canada

19. Multiple Reoperative Coronary Artery Bypass Surgery
Kevin D. Accola, Joseph M. Craver, Ellis L. Jones, William S. Weintraub, and Robert A. Guyton, Atlanta, GA

20. Synchronous Surgical Treatment of Ischemic Heart and Cerebrovascular Obstructive Disease. Long-Term Follow-up
Freddy Vermeulen, Ruben Hamerlijnck, Jo Defauw, and Sjef Ernst, Nieuwegein, Netherlands

21. Extracorporeal Circulation, Profound Hypothermia, and Circulatory Arrest for Otherwise Inoperable Intracranial Tumors
Mark D. Williams, W. Gerald Rainer, Henry Fieger, and Mary Sanchez, Denver, CO

22. Early Experience With Adult ECMO in the Modern Era
Harry L. Anderson III, Ralph E. Delius, James M. Sinard, Ken R. McCurry, Charles J. Shanley, Robin A. Chapman, Michael B. Shapiro, Jorge L. Rodriguez, and Robert H. Bartlett, Ann Arbor, MI

3:30–5:00 Business Meeting (members only)

Evening

8:00–10:00 **Surgical Motion Pictures**
Moderators: Alexander S. Geha, Cleveland, OH, and Robert A. Guyton, Atlanta, GA

1. Spiral Vein Graft for Superior Vena Cava Syndrome
George B. Haasler, Milwaukee, WI

2. Transcervical-Transsternal Maximal Thymectomy for Myasthenia Gravis
Alfred Jaretzki III, New York, NY

3. Correction of Postpneumonectomy Mediastinal Overshift by Insertion of Silastic Tissue Expander
Frederick L. Grover, J. Martin Smith, John H. Calhoon, William C. Hall, Deborah K. Rasch, Bruce M. Schnapf, David Cohen, Donald Campbell, and J. Kent Trinkle, San Antonio, TX

4. Aortic Valve Replacement With Pulmonary Autotransplant and Pulmonary Homograft Replacement for Rheumatic Aortic Insufficiency With Previous Mitral Repair in a 17-Year-Old Girl
Stephen B. Colvin, Frank C. Spencer, Aubrey Galloway, and Alan Katz, New York, NY

5. Warm Heart Surgery for Coronary Artery Bypass
Samuel V. Lichtenstein and Tomas A. Salerno, Toronto, Ont, Canada

6. Carinal Resection
Tsuguo Naruke, Tokyo, Japan

7. Perinatal Management of Massive Left Ventricular Rhabdomyoma
Harold D. Head, Kenneth L. Jue, and James V. Prochazka, Dayton, OH

8. Surgical Treatment of Infected Coarctation Graft and Aortobronchial Fistula
Michael J. DaValle, Chicago, IL

9. Thoracoscopic Management of a Recurrent Spontaneous Pneumothorax
J. K. Champion, Cleveland, TN

February 19, 1991 (Tuesday)
Morning

7:00–8:00 **Breakfast Sessions**
No advance registration. Attendance by ticket only and limited to 35 per seminar. Tickets can be purchased at registration desk on Monday, February 18, starting at 12:30 PM. Price of ticket covers attendance at seminar and continental breakfast. These sessions run concurrently.

CPT Nomenclature and Coding Workshop
Coding Clinic: Questions and Answers Session will focus on the cardiothoracic CPT and ICD coding process
Sidney Levitsky, Boston, MA

Problem Case Presentations (see pp 1032–4 for program)

8:15–11:15 **Scientific Session**

23. Mitral Valve Repair for Ischemic Mitral Insufficiency
William Hendren, James J. Nemec, Bruce W. Lytle, Floyd D. Loop, Paul C. Taylor,
Robert W. Stewart, and Delos M. Cosgrove III, Cleveland, OH

24. The Edwards Pericardial Aortic Valve: Intermediate Results
Robert W. M. Frater, Neal W. Salomon, W. Gerald Rainer, and Delos M. Cosgrove III, Bronx, NY

25. Operative Risk and Long-Term Results of Surgery for Left Ventricular Aneurysm
Masashi Komeda, Azhar Malik, Joan Ivanov, and Tirone E. David, Toronto, Ont, Canada

26. Angioscopic Evaluation of Intravascular Morphology After Coronary Endarterectomy
Bruce E. Keogh, Benjamin P. Bidstrup, Kenneth M. Taylor, and Ralph N. Sapsford, London, England

27. Total Cavopulmonary Anastomosis Versus the Conventional Modified Fontan Procedure
Jeffrey M. Pearl, Hillel Laks, Darryl G. Stein, Davis C. Drinkwater, Avedis Meneshian,
Isabel K. Hartmann, Barbara L. George, and Roberta G. Williams, Los Angeles, CA

28. Current Treatment for Wolff-Parkinson-White Syndrome: Results and Surgical Implications
Steven F. Bolling, Fred Morady, Hugh Calkins, Alan Kadish, Michael DeBuitleir, Macdonald Dick,
and Edward L. Bove, Ann Arbor, MI

29. Surgical Therapy of Paroxysmal Atrial Fibrillation Using the "Corridor" Operation
Jo Defauw, Gerard Guiraudon, Norbert van Hemel, Herre Kingma, Henry van Swieten,
Jacques de Bakker, and Freddy Vermeulen, Nieuwegein, Netherlands

11:15–12:00 **First Ralph D. Alley Lecture**

The Future of American Thoracic Surgery
Harry Schwartz, PhD, Scarsdale, NY
Columnist for *American Medical News*

Afternoon

1:30–5:00 **Parallel Scientific Sessions**
Session I

1:30–2:45 **General Thoracic II**
Moderator: Peter C. Pairolero, Rochester, MN

30. Acquired Nonmalignant Tracheoesophageal Fistula
Douglas J. Mathisen, Hermes C. Grillo, John C. Wain, Jr, and Alan D. Hilgenberg, Boston, MA

31. Combined Parietal Cell Vagotomy and Collis-Nissen Fundoplication
Rodney J. Landreneau, James M. Marshall, Joel A. Johnson, Stephen R. Hazelrigg,
Theresa M. Boley, and Jack J. Curtis, Columbia, MO

32. Clinical Spectrum of Bronchogenic Cysts of the Mediastinum and Lung in the Adult
Regent St-Georges, Jean Deslauriers, and Andre Duranceau, Ste-Foy, Que, Canada

33. Lung Cancer Model to Study Control of the Metastatic Process
William G. Hammond, Raymond L. Teplitz, and John R. Benfield, Sacramento, CA

34. Improved Management of the Paget-Schroetter Syndrome
Harold C. Urschel, Jr, and Maruf A. Razzuk, Dallas, TX

3:30–5:00 **Lung Transplant Research**

35. Normal Bronchial Healing Without Bronchial Wrapping in Preoperatively and Postoperatively Immunosuppressed Canine Single-Lung Transplantation
Joseph Auteri, Valluvan Jeevanandam, Juan Sanchez, Thomas Kirby, and Craig Smith, New York, NY

36. A Strategy to Increase the Pulmonary Donor Pool: The Use of Cadaver Lungs for Transplantation
Thomas M. Egan, C. Jake Lambert, Jr, Blair A. Keagy, and Benson R. Wilcox, Chapel Hill, NC

37. Prolonged Preservation of Dog Lung Allografts: The Role of Prostaglandins
R. J. Novick, K. R. Reid, L. Denning, J. Duplan, A. H. Menkis, and F. N. McKenzie, London, Ont, Canada

38. Improved Lung Preservation Using UW Solution Compared With Modified Euro-Collins Solution
Stephan Hirt, Thorsten Wahlers, Michael Jurmann, Lutz Dammenhayn, and Axel Haverich, Hannover, Germany

39. Neutrophils Are Not Necessary for Ischemia-Reperfusion Lung Injury
Cynthia N. Steimle, Steven F. Bolling, and G. Michael Deeb, Ann Arbor, MI

40. Pulmonary Granulocyte Sequestration: A Contributor to Pulmonary Injury and Early Failure of the Autoperfused Working Heart-Lung Preparation
Christopher M. Genco, James T. Diehl, Raymond J. Connolly, Karen Ramberg, Eugene Bernstein, Myron B. Peterson, Xi Zhang, and Richard J. Cleveland, Boston, MA

1:30–5:00 **Session II**

1:30–2:45 **Cardiac Research**
Moderator: Timothy J. Gardner, Baltimore, MD

41. Myocardial Oxygen Consumption During Right Heart Bypass
Matthew S. Coons, Thomas T. Woloszyn, Thierry A. Folliguet, Clem Rodriguez, Samir Basu, Joseph N. Cunningham, Jr, and Corrado P. Marini, Brooklyn, NY

42. Recovery of Myocardial Oxygen Utilization Efficiency in Stunned Myocardium
Satoshi Furukawa, Gerhard Kreiner, Joseph Bavaria, James Streicher, and L. Henry Edmunds, Jr, Philadelphia, PA

43. Determinants of Myocardial Oxygen Consumption in Dilated Cardiomyopathy Influenced by Epinephrine Administration
Cornelius Dyke, K. Francis Lee, Jitendra Parmar, Rebecca Dignan, Thomas Yeh, Jr, Anwar Abd-Elfattah, and Andrew S. Wechsler, Richmond, VA

44. Resuscitation of Injured Myocardium With Adenosine and Biventricular Assist
Todd L. Demmy, James A. Magovern, Race L. Kao, and George J. Magovern, Pittsburgh, PA

45. The Effects of Flow Rate and Temperature on Regional Blood Flow and Oxygen Consumption During Cardiopulmonary Bypass in a Canine Model
W. Douglas Lazenby, Wilson Ko, John A. Zelano, Nathan Lebowitz, Yong T. Shin, O. Wayne Isom, and Karl H. Krieger, New York, NY

3:30–5:00 **Electrophysiology and Cardiac Research**
Moderator: Robert A. Guyton, Atlanta, GA

46. Selective Surgical Approach for Atrioventricular Reentrant Tachycardia
Alexander S. Geha, Lee A. Biblo, Mark D. Carlson, and Albert L. Waldo, Cleveland, OH

47. Effect of Location of AICD Patch Electrodes on Left Ventricular Diastolic Pressure-Volume Curve in Pigs
Joseph S. Auteri, Valluvan Jeevanandam, Juan A. Sanchez, and Henry M. Spotnitz, New York, NY

48. A New Extraaortic Counterpulsation Device
John A. Zelano, Wilson Ko, Richard Lazzaro, W. Douglas Lazenby, Stephen Topaz, Willem J. Kolff, O. Wayne Isom, and Karl H. Krieger, New York, NY and Salt Lake City, UT

49. Retrograde Cerebral Perfusion Through a Superior Vena Cava Cannula Protects the Brain
Akihiko Usui, Toshiro Hotta, Manabu Hiroura, Mitsuya Murase, Masanobu Maeda, Takashi Watanabe, Eiji Takeuchi, Minoru Tanaka, and Toshio Abe, Nagoya, Japan

50. Calcification of Porcine Valves: A Successful New Method of Antimineralization
John Parker Gott, Pan-Chih, Lynne M. A. Dorsey, John L. Jay, Frederick J. Schoen, and Robert A. Guyton, Atlanta, GA

51. Effects of Internal Mammary Artery Dissection on Phrenic Nerve Function and Perfusion
James W. O'Brien, Scott H. Johnson, Scott J. VanSteyn, J. Scott Kabas, Robbin E. Sharpe, and Peter K. Smith, Durham, NC

1:30–5:00 **Session III**

1:30–2:45 **Adult Cardiac II**
Moderator: Delos M. Cosgrove III, Cleveland, OH

52. Improvement in Surgery for Acute Type A Aortic Dissection. Intraluminal Graft, Dacron Prosthesis, or Biological Glue?
J. R. Seguin, J.-M. Frappier, and P. A. Chaptal, Montpellier, France

53. Long-Term Follow-up of Descending Aortic Dissection
John Elefteriades, James Hartleroad, John Baldwin, Richard Gusberg, Henry Black, Gary Kopf, Kenneth Franco, Michael Dewar, and Graeme Hammond, New Haven, CT

54. Advances in the Surgery of the Aortic Arch
Joachim Laas, Michael Jurmann, Markus Heinemann, and Hans Georg Borst, Hannover, Germany

55. A New Extended Vertical Transseptal Atrial Approach for Mitral Valve Surgery in 36 Patients
Gerard M. Guiraudon, Raj Kaushik, John Ofiesh, and Douglas G. McLellan, London, Ont, Canada

56. Prospective Study Using Intraoperative Transesophageal Echocardiography to Grade Atheromatous Aortic Arch Disease: Early Results
Greg H. Ribakove, Edward S. Katz, Aubrey C. Galloway, Rick A. Esposito, Itzhak Kronzon, and Frank C. Spencer, New York, NY

3:30–5:00 **Congenital Cardiac**
Moderator: Adnan Cobanoglu, Portland, OR

57. The Use of an Experimental "One-Way Valved ASD Patch": Animal Studies and Preliminary Clinical Experience
J. Barak and B. A. Vidne, Tel Aviv, Israel

58. The Partial Fontan: Advantages of an Adjustable Interatrial Communication
Jeffrey M. Pearl, Hillel Laks, Davis C. Drinkwater, Darryl G. Stein, Barbara George, and Roberta Williams, Los Angeles, CA

59. Successful Relief of Neonatal Congenital Aortic Stenosis Utilizing Inflow Occlusion
Michael A. Grosso, John St. Cyr, David Fullerton, David N. Campbell, and David R. Clarke, Denver, CO

60. Options for Surgical Treatment of Subaortic Stenosis in the Univentricular Heart
Tom R. Karl, Kevin Watterson, Shunji Sano, and Roger B. B. Mee, Melbourne, Australia

61. Surgical Options in Subaortic Stenosis Associated With Endocardial Cushion Defects
Serafin DeLeon, Michel Ilbawi, William Wilson, Rene Arcilla, Otto Thilenius, Saroja Bharati, Maurice Lev, and Farouk Idriss, Chicago, IL

62. Repair of Truncus Arteriosus in Infancy
Jeffrey M. Pearl, Hillel Laks, Davis Drinkwater, Orrin Ailloni-Charas, Frank Giacobetti, Barbara George, and Roberta Williams, Los Angeles, CA

Evening

7:00 **Reception for Members and Guests**

February 20, 1991 (Wednesday)
Morning

7:00–8:00 **Breakfast Sessions**
No advance registration. Attendance by ticket only and limited to 35 per seminar. Tickets can be purchased at registration desk on Monday, February 18, starting at 12:30 PM. Price of ticket covers attendance at seminar and continental breakfast. These sessions run concurrently.

CPT Nomenclature and Coding Workshop
Coding Clinic: Questions and Answers Session will focus on the cardiothoracic CPT and ICD coding process
Sidney Levitsky, Boston, MA

Clinical Workshops (see p 1034 for program)

8:15–12:00 **Scientific Session**

63. Laryngotracheoplastic Resection for Subglottic Stenosis: Results of Excision and Primary Reconstruction
Hermes C. Grillo, Douglas J. Mathisen, and John C. Wain, Jr, Boston, MA

64. Surgical Management of Medically Unresponsive Pulmonary Infection With *Mycobacterium tuberculosis* and Other Mycobacterial Organisms
Marvin Pomerantz, Marian Goble, Lori Madsen, and Michael Iseman, Denver, CO

65. Malignant Esophagorespiratory Fistula: Treatment Options and Survival
Michael Burt, William Diehl, Manjit S. Bains, Robert J. Ginsberg, Nael Martini, Patricia M. McCormack, and Valerie W. Rusch, New York, NY

66. Early Experience of Double Lung Transplantation for Cystic Fibrosis
Hani Shennib, Michel Noirclerc, and the Cystic Fibrosis Transplant Study Group, Montreal, Que, Canada

67. Intraoperative Aortic Dissection
Robert J. Still, Alan Hilgenberg, Cary W. Akins, Mortimer J. Buckley, and Willard M. Daggett, Boston, MA

68. Results of Surgery for Recent Myocardial Infarction Using Warm Aerobic Arrest
Samuel V. Lichtenstein and Tomas A. Salerno, Toronto, Ont, Canada

69. Experience With Surgical Repair of Aortic Root Aneurysms: Results in 280 Patients
Clifton T. P. Lewis, Denton A. Cooley, Oscar Talledo, and Michael C. Murphy, Houston, TX

70. Blunt Injuries of the Thoracic Aorta
Alan D. Hilgenberg, Diana L. Logan, Cary W. Akins, Mortimer J. Buckley, Willard M. Daggett, Gus J. Vlahakes, and David F. Torchiana, Boston, MA

71. Bilateral Latissimus Dorsi Cardiomyoplasty: A New Procedure for Biventricular Hemodynamic Augmentation
James A. Magovern, Anthony P. Furnary, Ignacio Y. Christlieb, Race L. Kao, and George J. Magovern, Pittsburgh, PA

72. Successful Transplantation of Hearts Harvested 30 Minutes After Death From Exsanguination
Steven R. Gundry, Javier Alonso de Begona, Motohiro Kawauchi, and Leonard L. Bailey, Loma Linda, CA

February 19, 1991 (Tuesday morning)

7:00–8:00 PROBLEM CASE PRESENTATIONS
(10 Simultaneous Sessions)
Session Committee: Delos M. Cosgrove III, Cleveland, OH, and Timothy J. Gardner, Baltimore, MD

SEMINAR I
DIFFICULT CARDIAC REPAIRS
Discussion Leader: Cary W. Akins, Boston, MA

1. A Large Pseudoaneurysm of the Left Ventricle Associated With Mitral Regurgitation
Mohamed Aziz, Ibrahim Abdelmeguid, and Armand Piwnica, Paris, France

2. Mitral Valve Replacement in a Patient With Idiopathic Thrombocytopenic Purpura
Kenneth Richards and Victor A. Ferraris, Albany, NY

3. Acute Myocardial Infarction and Ventricular Septal Defect
Idris Ali, Stephen E. Fremes, George T. Christakis, and Bernard S. Goldman, Toronto, Ont, Canada

SEMINAR II
CARDIAC INFECTIONS
Discussion Leader: Hartzell V. Schaff, Rochester, MN

4. *Staphylococcus aureus* Endocarditis With Pulmonary Edema, Cerebral and Systemic Embolization in a 26-Year-Old Man
Hugh Scully, Xavier Mousset, John Mullen, and Paul Petrasek, Toronto, Ont, Canada

5. Infective Endocarditis in Patients With Left Ventricular Aneurysm
Azhar Malik, Masashi Komeda, and Tirone E. David, Toronto, Ont, Canada

6. Bioprosthetic Valve Endocarditis
George T. Christakis, Idris Ali, Stephen E. Fremes, and Bernard S. Goldman, Toronto, Ont, Canada

SEMINAR III
CORONARY
Discussion Leader: Ellis L. Jones, Atlanta, GA

7. A 7-cm Ruptured Aneurysm of the Right Coronary Artery
R. Vijayanagar, Paul F. Eckstein, Rafael Salas, Marshall DeSantis, Raymond S. Waters, and Akshay Desai, Tampa, FL

8. Unrecognized Coronary Ostial Stenosis
Richard Bloom, Idris Ali, George Christakis, and Bernard Goldman, Toronto, Ont, Canada

9. Acute Cardiovascular Collapse After Routine CABG: Possible Protamine Sulfate Hypersensitivity in a Young Man With Vasectomy 10 Years Before
Sam Hunter and Arlen Holter, Minneapolis, MN

SEMINAR IV
GREAT VESSELS
Discussion Leader: Randall B. Griepp, New York, NY

10. Acute Ascending Aortic Dissection Involving Right Coronary Artery
Joseph S. Coselli, Houston, TX

11. Ascending Aortic Dissection During Coronary Revascularization
Richard M. Engelman, John A. Rousou, Joseph E. Flack III, and David W. Deaton, Springfield, MA

12. Pseudoaneurysm of the Innominate Artery
Julian Belisle, James T. Diehl, Richard J. Cleveland, John Waciuma, and Lynn Welchel, Boston, MA

SEMINAR V
CONGENITAL
Discussion Leader: Kevin Turley, San Francisco, CA

13. Pulmonary Venous Obstruction After Repair of Total Anomalous Pulmonary Venous Connection
Christo I. Tchervenkov, Jerome B. Riebman, Marie J. Beland, and Anthony R. C. Dobell, Montreal, Que, Canada

14. Ebstein's Anomaly, VSD and Pulmonary Hypertension Presenting in Infancy
Tom R. Karl and Roger B. B. Mee, Melbourne, Australia

15. Isolated Unilateral Pulmonary Artery Agenesis and Necrotizing Pneumonia in a Neonate
Charles C. Canver, John D. Pigott, and Robert M. Mentzer, Jr, Buffalo, NY

SEMINAR VI
AIRWAYS
Discussion Leader: Thomas R. J. Todd, Ottawa, Ont, Canada

16. Treatment of Diffuse Tracheomalacia
George B. Haasler, Milwaukee, WI

17. Tracheal Complication of Transhiatal Esophagectomy
L. A. Gorenstein, J. G. Abel, and G. A. Patterson, Toronto, Ont, Canada

18. Complex Rupture of Major Airways
A. G. Justicz and P. N. Symbas, Atlanta, GA

SEMINAR VII
CHEST WALL AND MEDIASTINUM
Discussion Leader: Victor F. Trastek, Rochester, MN

19. Anterior Superior Mediastinal Mass With Superior Vena Cava Syndrome
Warren A. Williamson, John M. Streitz, Jr, and David M. Shahian, Burlington, MA

20. Lymphangiectasis of the Mediastinum With Chylothorax and Chylopericardium
Himmet Dajee, John Eilbert, Viney Soni, and Glen Justice, Newport Beach, CA

21. Chest Wall Tumor of Fifth Rib Posteriorly
C. Frederick Kittle, Chicago, IL

SEMINAR VIII
ESOPHAGUS
Discussion Leader: Mark K. Ferguson, Chicago, IL

22. Drainage and Irrigation Technique as a Primary Treatment for Late Presentation of Spontaneous Esophageal Rupture
Shlomo Gabbay, Nezih Ozdemir, Robert J. Wilcott, and James N. Heller, Newark, NJ

23. Management of Esophagopulmonary Fistula in a Patient With Megaesophagus Secondary to Achalasia
David G. Kitts, George E. Cimochowski, Jeffrey C. Milliken, and Russell A. Williams, Torrance, CA

24. Esophagopericardial Fistula Four Years After Radiotherapy for Esophageal Cancer
Paul F. Waters, Los Angeles, CA

SEMINAR IX
PULMONARY 1
Discussion Leader: Larry R. Kaiser, St. Louis, MO

25. Surgical Management of Aspergilloma and Hemoptysis
J. W. Randolph Bolton and Harold L. Lazar, Boston, MA

26. Massive Wheat Grain Aspiration—A Survivor
W. T. Kidd and P. E. Blundell, Montreal, Que, Canada

27. Complications of Group A Streptoccocus Pneumonia
Michael P. Koumjian and Lucien N. Jassy, LaMesa, CA

SEMINAR X
PULMONARY 2
Discussion Leader: Thomas Rice, Cleveland, OH

28. Postpneumonectomy Tension Chylothorax
Julian Belisle, Irving Madoff, and James T. Diehl, Boston, MA

29. Bronchogenic Carcinoma With Empyema
Darryl S. Weiman, L. Michael Graver, and Denis H. Tyras, New Hyde Park, NY

30. Late Complication of Plombage
Michael D. Horowitz and Richard J. Thurer, Miami, FL

Wednesday, February 20, 1991

7:00–8:00 CLINICAL WORKSHOPS
(10 Simultaneous Sessions)
Session Committee: Adnan Cobanoglu, Portland, OR, and Douglas J. Mathisen, Boston, MA

WORKSHOP I
MANAGEMENT OF STAGE III LUNG CANCER
Alex G. Little, Las Vegas, NV

WORKSHOP II
TRACHEOBRONCHIAL TRAUMA
Jean Deslauriers, Ste-Foy, Que, Canada

WORKSHOP III
MANAGEMENT OF MEDIASTINAL MALIGNANCIES
Robert J. Ginsberg, New York, NY

WORKSHOP IV
TECHNICAL CONSIDERATIONS OF ESOPHAGEAL SUBSTITUTES
Richard F. Heitmiller, Baltimore, MD

WORKSHOP V

ALTERNATIVE CONDUITS FOR CORONARY REVASCULARIZATION
Noel L. Mills, New Orleans, LA

WORKSHOP VI

OPERATIVE INTERVENTION IN ATRIAL FIBRILLATION
James L. Cox, St. Louis, MO

WORKSHOP VII

MITRAL VALVE REPAIR
Steven B. Colvin, New York, NY

WORKSHOP VIII

REPEAT OPERATION FOR CORONARY REVASCULARIZATION
Bruce W. Lytle, Cleveland, OH

WORKSHOP IX

HOMOGRAFT USE IN INFANTS AND CHILDREN
John J. Lamberti, San Diego, CA

WORKSHOP X

THE ARTERIAL SWITCH OPERATION
William G. Williams, Toronto, Ont, Canada

Author Index
Volume 50 (July–December 1990)

KEY TO ABBREVIATIONS USED FOR ARTICLE CLASSIFICATION

* = Original Article
AN = Announcement
CL = Classics in Thoracic Surgery
CR = Case Report

CP = Correspondence
CT = Collective Review
CV = Current Review
DI = Discussion
ED = Editorial
HW = How to Do It

KR = Key References
PA = Presidential Address
RE = Report
RV = Review of Recent Books

Aaron BL, Alyono D. Ventricular perforation with valvoplasty. 1990;50:1022,CP

Abbruzzese PA, Napoleone A, Bini RM, Annecchino FP, Merlo M, Parenzan L. Valve insufficiency after atrioventricular septal defect repair: differences between patients with and without Down's syndrome? Reply. 1990;50:854,CP

Abel RM. *see* McCullough, Abel, & others. 1990;50:663–4,CR

Abt AB. *see* Haluck, Richenbacher, & others. 1990;50:392–5*

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Adkins MS, Raccuia JS, Acinapura AJ. Esophageal perforation in a patient with acquired immunodeficiency syndrome. 1990;50:299–300,CR

Adler RH. Collis gastroplasty: origin and evolution. 1990;50:839–42,CL

Ahmed S. *see* Shapira, Zabatino, & others. 1990;50:268–73*

Ahn C, Harvey JC. Mediastinal hibernoma, a rare tumor. 1990;50:828–30,CR

Airan B, Sharma R, Iyer KS, Kalia PK, Singh MK, Shrivastava S, Rao IM. Malignant thymoma presenting as intracardiac tumor and superior vena caval obstruction. 1990;50:989–91,CR

Aizawa Y. *see* Miyamura, Nakagomi, & others. 1990;50:476–8,CR

Akçevin A. *see* Bakay, Akçevin, & others. 1990;50:553–6*

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Albes J, Haverich A, Freihorst J, von der Hardt H, Manthey-Stiers F. Management of mycotic rupture of the ascending aorta after heart-lung transplantation. 1990;50:982–3,CR

Albrechtsen OK. *see* Lund, Pilegaard, & others. 1990;50:396–406*

Alexander JA. *see* Epstein, Duncan, & others. 1990;50:94–7*

Alexi-Meskhishvili V. Anomalous left coronary artery surgery. 1990;50:511–2,CP

Ali I, Unruh H. Management of empyema thoracis. 1990;50:355–9*

Ali IM, Butler CK, Armour JA, Murphy DA. Modification of supraventricular tachyarrhythmias by stimulating atrial neurons. 1990;50:251–6*

Alpern JB. *see* Kolff, McClurken, & others. 1990;50:512,CP

Altorki NK. Esophagectomy for Barrett's esophagus. Reply. 1990;50:858–9,CP

Alyono D. *see* Aaron & Alyono. 1990;50:1022,CP

Amodeo A, Corno A, Marino B, Carta MG, Marcelletti C. Combined repair of transposed great arteries and total anomalous pulmonary venous connection. 1990;50:820–1,CR

Anderson KC. *see* Goodnough, Johnston, & others. 1990;50:675–83,CV

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Angelini GD, Azzu AA. A fiber-optic retractor for harvesting the internal mammary artery. 1990;50:314–5,HW

Angelini GD. *see* Bryan & Angelini. 1990;50:330,CP

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Antunes MJ. Clinical performance of St. Jude and Medtronic-Hall prostheses: a randomized comparative study. 1990;50:743–7*

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Astudillo J, Conill C. Role of postoperative radiation therapy in stage IIIa non-small cell lung cancer. 1990;50:618–23*

Atsumi N. *see* Sakakibara, Hori, & others. 1990;50:857–8,CP

Attar S, Hankins JR, Suter CM, Coughlin TR, Sequeira A, McLaughlin JS. Esophageal perforation: a therapeutic challenge. 1990;50:45–51*

Attar S. *see* Daniel, Tribble, & others. 1990;50:186–9,DI

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Azariades M, Fessler CL, Floten HS, Starr A. Five-year results of coronary bypass grafting for patients older than 70 years: role of internal mammary artery. 1990;50:940–5*
Azzu AA. *see* Angelini & Azzu. 1990;50:314–5,HW

Babu SC. *see* Moggio, Praeger, & others. 1990;50:146–8,HW
Baciewicz PA, del Rio C, Goncalves MA, Lattouf OM, Guyton RA, Morris DC. Catastrophic thrombosis of porcine aortic bioprostheses. 1990;50:817–9,CR
Backer CL, Idriss FS, Zales VR, Mavroudis C. Cardiac transplantation for hypoplastic left heart syndrome: a modified technique. 1990;50:894–8*
Bahnson HT. *see* Keenan, Armitage, & others. 1990;50:748–53*
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Bakay C, Akçevin A, Süzer K, Paker T, Türkoğlu H, Akpinar B, Aytaç A, Demiroğlu C. Combined internal mammary artery graft for coronary artery revascularization. 1990;50: 553–6*
Baker JE, Boerboom LE, Olinger GN. Cardioplegia-induced damage to ischemic immature myocardium is independent of oxygen availability. 1990;50:934–9*
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Barratt-Boyes BG. Choreoathetosis as a complication of cardiopulmonary bypass. 1990;50:693–4,ED
Bartlett RH. *see* Delius, Zwischenberger, & others. 1990;50:791–5*
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Baumgartner F, Sheppard B, de Virgilio C, Esrig B, Harrier D, Nelson RJ, Robertson JM. Tracheal and main bronchial disruptions after blunt chest trauma: presentation and management. 1990;50:569–74*
Baumgartner WA. *see* Pillai, Bando, & others. 1990;50:211–4*
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Baxter R, McFadden PM, Gradman M, Wright A. Scimitar syndrome: cine magnetic resonance imaging demonstration of anomalous pulmonary venous drainage. 1990;50: 121–3,CR
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Benfield JR. Paraplegia associated with the use of oxidized cellulose in posterolateral thoracotomy incisions. Invited commentary. 1990;50:290*
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Bevelaqua F, Garritan S, Haas F, Salazar-Schicchi J, Axen K, Reggiani J-L. Complications after cardiac operations in patients with severe pulmonary impairment. 1990;50: 602–6*
Bhattacharyya SS, Trivedi A, Pendkar R, Thacker JJ. Hemostatic technique for internal mammary artery anastomotic bleeding. 1990;50:316–7,HW
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Blakeman BP, Sullivan HJ, Foy BK, Sobotka PA, Pifarre R. Internal mammary artery revascularization in the patient on long-term renal dialysis. 1990;50:776–8*
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Bolling SF, Kirsh MM. Alternate method for revascularization of the posterior lateral coronary artery. 1990;50:838,HW
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KEY TO ABBREVIATIONS USED FOR ARTICLE CLASSIFICATION

* = Original Article
AN = Announcement
CL = Classics in Thoracic Surgery
CR = Case Report
CP = Correspondence
CT = Collective Review
CV = Current Review
ED = Editorial
HW = How to Do It
KR = Key References
PA = Presidential Address
RE = Report
RV = Review of Recent Books

Ablation
Surgical epicardial ablation of left ventricular pathway using sling exposure (Guiraudon, Klein, & others) 1990;50:968–71*
Abnormalities
Cardiac transplantation for hypoplastic left heart syndrome: a modified technique (Backer, Idriss, & others) 1990;50:894–8*
Cardiac transplantation for neonates with hypoplastic left heart syndrome (Mayer) 1990;50:864–5,ED
Congenital tracheoesophageal fistula associated with carcinoma of the lung in an adult (Campbell & Salisbury) 1990;50:978–9,CR
Late results of aortic valvotomy for congenital valvar aortic stenosis (DeBoer, Robbins, & others) 1990;50:69–73*
Left ventricular outflow tract obstruction with aortic arch anomalies and ventricular septal defect (Sell) 1990;50:854–5,CP; Reply (Iwahara, Ino, & Yabuta) 1990;50:855,CP
Value of transesophageal echocardiography during repair of congenital heart defects (Dan, Bonato, & others) 1990;50:637–43*
Abscess
Job's syndrome: a rare cause of recurrent lung abscess in childhood (Lui & Inculet) 1990;50:992–4,CR
Acquired immunodeficiency syndrome
Esophageal perforation in a patient with acquired immunodeficiency syndrome (Adkins, Raccuia, & Acinapura) 1990;50:299–300,CR
Tuberculosis (Pomerantz) 1990;50:498–9,KR
ACTH syndrome, ectopic. *See* Neoplastic endocrine-like syndromes.
Adolescent
Prolonged extracorporeal life support of pediatric and adolescent cardiac transplant patients (Delius, Zwischenberger, & others) 1990;50:791–5*
Age factors
Five-year results of coronary bypass grafting for patients older than 70 years: role of internal mammary artery (Azariades, Fessler, & others) 1990;50:940–5*
Long-term results of operation for non–small cell lung cancer in the elderly (Ishida, Yokoyama, & others) 1990;50:919–22*
Severe aortic stenosis in octogenarians: is operation an acceptable alternative? (Deleuze, Loisance, & others) 1990;50:226–9*
Uncomfortable issues (Edmunds) 1990;50:173–4,ED
Air
Air contamination in open heart surgery with disposable coveralls, gowns, and drapes (Verkkala, Mäkelä, & others) 1990;50:757–61*
Airway obstruction
Endoscopic relief of airway obstruction (Cavaliere & Ardigò) 1990;50:163,CP; Reply (Mathisen & Grillo) 1990;50:164,CP
Amiodarone
Long-term amiodarone administration protects against global myocardial ischemia (Karlson, Hopkins, & others) 1990;50:575–8*
Analgesia
Continuous epidural hydromorphone for postthoracotomy pain relief (Brodsky, Chaplan, & others) 1990;50:888–93*
Epidural analgesia: different strokes for different folks (Faber) 1990;50:862–3,ED
Anastomosis
Excision of the aortic wall in the surgical treatment of acute type-A aortic dissection (Massimo, Presenti, & others) 1990;50:274–6*
Hemostatic technique for internal mammary artery anastomotic bleeding (Bhattacharyya, Trivedi, & others) 1990;50:316–7,HW
Liver transplantation with atrioatrial anastomosis for Budd-Chiari syndrome (Carrel, Decurtins, & others) 1990;50:658–60,CR
Results of aortic anastomoses made under tension using polydioxanone suture (Haluck, Richenbacher, & others) 1990;50:392–5*
Aneurysm
Circumflex artery ventricular fistula and pseudoaneurysm after mitral reoperation (Sutter, Goldman, & others) 1990;50:826–7,CR
Mid-descending aortic traumatic aneurysms (Rabinsky, Sidhu, & Wagner) 1990;50:155–60,CT
Morphological and histopathological aspects of aneurysms after patch aortoplasty for coarctation (Heikkinen, Sariola, & others) 1990;50:946–8*
Presumed Kawasaki disease resulting in multiple coronary artery aneurysms in an adult (Brevard & Smith) 1990;50:291–3,CR
Successful surgical treatment of incessant automatic atrial tachycardia with atrial aneurysm (Miyamura, Nakagomi, & others) 1990;50:476–8,CR
Swan-Ganz catheter-induced massive hemoptysis and pulmonary artery false aneurysm (Feng, Singh, & others) 1990;50:644–6,CR
Systemic venous aneurysms (Yokomise, Nakayama, & others) 1990;50:460–2,CR

July issue, pp 1–172; August issue, pp 173–342; September issue, pp 343–518; October issue, pp 519–688; November issue, pp 689–860; December issue, pp 861–1076.

Angina
- Internal mammary artery graft to pulmonary vasculature fistula: a cause of recurrent angina (Johnson, Schmaltz, & others) 1990;50:297–8,CR
- Steal phenomenon caused by a parallel branch of the internal mammary artery (Schmid, Heublein, & others) 1990;50:463–4,CR

Angiography
- Systemic venous aneurysms (Yokomise, Nakayama, & others) 1990;50:460–2,CR

Angioplasty, transluminal, percutaneous coronary
- Surgical standby for coronary angioplasty (Ullyot) 1990;50:3–4,ED
- Surgical standby for percutaneous transluminal coronary angioplasty: a survey of patterns of practice (Cameron, Stinson, & others) 1990;50:35–9*

Antibiotics
- Antimicrobial prophylaxis for open heart operations (Miedzinski, Callaghan, & others) 1990;50:800–7*
- Treatment of postoperative infection of ascending aorta and transverse aortic arch, including use of viable omentum and muscle flaps (Coselli, Crawford, & others) 1990;50:868–81*

Aorta
- Aortic dissection with the entrance tear in transverse aorta: analysis of 12 autopsy patients (Roberts & Roberts) 1990;50:762–6*
- Aortobronchial fistula after an aortic operation (Ishizaki, Tada, & others) 1990;50:975–7,CR
- Easy cardioplegia delivery, aortic venting, and reperfusion technique (Kauten) 1990;50:833–5,HW
- Excision of the aortic wall in the surgical treatment of acute type-A aortic dissection (Massimo, Presenti, & others) 1990;50:274–6*
- Intraoperative ultrasonic imaging of the ascending aorta in ischemic heart disease (Ohtaki, Itoh, & others) 1990;50:539–42*
- Management of aortobronchial fistula with graft replacement and omentopexy (Paull & Keagy) 1990;50:972–4,CR
- Management of mycotic rupture of the ascending aorta after heart-lung transplantation (Albes, Haverich, & others) 1990;50:982–3,CR
- Medical management of acute traumatic rupture of the aorta (Walker & Pate) 1990;50:965–7*
- Mid-descending aortic traumatic aneurysms (Rabinsky, Sidhu, & Wagner) 1990;50:155–60,CT
- Prevention of paraplegia during aortic operations (Wadouh, Wadouh, & others) 1990;50:543–52*
- Results of aortic anastomoses made under tension using polydioxanone suture (Haluck, Richenbacher, & others) 1990;50:392–5*
- Sutureless ring graft replacement of ascending aorta and aortic arch (Oz, Ashton, & others) 1990;50:74–9*
- Thoracic spinal fractures and aortic rupture: a significant and fatal association (Sturm, Hines, & Perry) 1990;50:931–3*
- Treatment of postoperative infection of ascending aorta and transverse aortic arch, including use of viable omentum and muscle flaps (Coselli, Crawford, & others) 1990;50:868–81*
- Use of the variable-length intraluminal sutureless graft (Graver & Tyras) 1990;50:624–30*

Aorta, thoracic
- Isolated left carotid artery in CHARGE association: diagnosis and repair (Ghalili, Issenberg, & others) 1990;50:130–2,CR
- Left ventricular outflow tract obstruction with aortic arch anomalies and ventricular septal defect (Sell) 1990;50:854–5,CP; Reply (Iwahara, Ino, & Yabuta) 1990;50:855,CP
- Malignant fibrous histiocytoma (Laas & Schmid) 1990;50:164, CP; Reply (Lazar) 1990;50:165,CP; (Craddock) 1990;50:165,CP
- Sutureless ring graft replacement of ascending aorta and aortic arch (Oz, Ashton, & others) 1990;50:74–9*

Aortic arch. *See* Aorta, thoracic.

Aortic coarctation
- Coarctation of the aorta (van Son) 1990;50:852–3,KR
- Morphological and histopathological aspects of aneurysms after patch aortoplasty for coarctation (Heikkinen, Sariola, & others) 1990;50:946–8*
- Paradoxical hypertension after repair of coarctation of the aorta: a review of its causes (Sealy) 1990;50:323–9,CV

Aortic valve
- Reversibility of pulmonary artery hypertension in aortic stenosis after aortic valve replacement (Tracy, Proctor, & Hizny) 1990;50:89–93*
- Severe aortic stenosis in octogenarians: is operation an acceptable alternative? (Deleuze, Loisance, & others) 1990;50:226–9*
- Timing of operation for aortic regurgitation: relation to postoperative contractile state (Taniguchi, Nakano, & others) 1990;50:779–85*
- Uncomfortable issues (Edmunds) 1990;50:173–4,ED

Aortic valve stenosis
- Aortic valve repair for aortic stenosis in adults (Shapira, Lemole, & others) 1990;50:110–20*
- Critical aortic stenosis in the first month of life: surgical results in 26 infants (Karl, Sano, & others) 1990;50:105–9*
- Late results of aortic valvotomy for congenital valvar aortic stenosis (DeBoer, Robbins, & others) 1990;50:69–73*
- Long-term prosthesis-related and sudden cardiac-related complications after valve replacement for aortic stenosis (Lund, Pilegaard, & others) 1990;50:396–406*
- Norwood operation for univentricular heart with subaortic stenosis in the neonate (Tchervenkov, Béland, & others) 1990;50:822–5,CR
- Reappraisal of localized resection for subvalvar aortic stenosis (Stewart, Merrill, & others) 1990;50:197–203*
- Repair of supravalvar aortic stenosis: cardiovascular morphometric and hemodynamic results (Braunstein, Sade, & others) 1990;50:700–7*
- Tubular supravalvar aortic stenosis: replacement of ascending aorta in a young patient (Gargiulo, Zannini, & others) 1990;50:467–8,CR

Aortocoronary bypass. *See* Coronary artery bypass.

Aortopexy
- Relief of tracheal compression by aortopexy (Clevenger, Othersen, & Smith) 1990;50:524–9*

Aprotinin
- How should the risk of bank blood transfusion in open heart operations be minimized? (Santoli, Scrofani, & Santoli) 1990;50:1021–2,CP

Arrhythmias
- Modification of supraventricular tachyarrhythmias by stimulating atrial neurons (Ali, Butler, & others) 1990;50:251–6*
- Prophylactic digitalization for thoracotomy: a reassessment (Ritchie, Bowe, & Gibbons) 1990;50:86–8*

Arterial switch operation
- New technique for the arterial switch operation in difficult situations (Takeuchi & Katogi) 1990;50:1000–1,HW

Arteries
- Alternate method for revascularization of the posterior lateral coronary artery (Bolling & Kirsh) 1990;50:838,HW
- Anomalous left coronary artery surgery (Alexi-Meskhishvili) 1990;50:511,CP; Reply (Clarke) 1990;50:512,CP
- Arteriosclerosis of the gastroepiploic and internal thoracic arteries (Suma & Takanashi) 1990;50:413–6*
- Choice of surgical clips for mammary artery take-down (Hovaguimian & Floten) 1990;50:856,CP; Reply (Gold) 1990;50: 856–7,CP
- Combined repair of transposed great arteries and total anomalous pulmonary venous connection (Amodeo, Corno, & others) 1990;50:820–1,CR
- Coronary revascularization in a child with Kawasaki disease: use of right gastroepiploic artery (Takeuchi, Gomi, & others) 1990;50:294–6,CR
- Free gastroepiploic artery graft (Mills & Everson) 1990;50: 858,CP
- Internal mammary artery bypass after the arterial switch operation (Rheuban, Kron, & Bulatovic) 1990;50:125–6,CR
- Internal mammary artery-to-pulmonary artery shunt in infants (Sakakibara, Hori, & others) 1990;50:857–8,CP
- Isolated left carotid artery in CHARGE association: diagnosis and repair (Ghalili, Issenberg, & others) 1990;50:130–2,CR
- Pharmacological response of internal mammary artery and gastroepiploic artery (Koike, Suma, & others) 1990;50: 384–6*
- Pleuropulmonary morbidity: internal thoracic artery versus saphenous vein graft (Hurlbut, Myers, & others) 1990;50: 959–64*
- Presumed Kawasaki disease resulting in multiple coronary artery aneurysms in an adult (Brevard & Smith) 1990;50: 291–3,CR
- Spasm of arterial conduits in aorta-coronary bypass grafting (van Son & Vincent) 1990;50:1019,CP; Reply (Suma) 1990; 50:1019–200,CP
- Systemic arterial air embolism in penetrating lung injury (Estrera, Pass, & Platt) 1990;50:257–61*

Arteriosclerosis
- Arteriosclerosis of the gastroepiploic and internal thoracic arteries (Suma & Takanashi) 1990;50:413–6*

Aspiration
- Intraoperative bronchial aspiration of ruptured pulmonary hydatid cysts (Saidi & Rezvan-Nobahar) 1990;50:631–6*

Aspirin
- Aspirin, blood loss, and transfusion (Gay) 1990;50:345,ED
- Blood conservation in cardiac surgery (Scott, Kessler, & Wernly) 1990;50:843–51,CV
- Low-dose preoperative aspirin therapy, postoperative blood loss, and transfusion requirements (Taggart, Siddiqui, & Wheatley) 1990;50:425–8*

Assisted circulation
- Clinical experience with portable cardiopulmonary bypass in cardiac arrest patients (Hartz, LoCicero, & others) 1990;50: 437–41*
- Effects of left ventricular assist for cardiogenic shock on cardiac function and organ blood flow distribution (Sukehiro & Flameng) 1990;50:374–83*
- A practical mechanical respirator, 1929: the "iron lung" (Meyer) 1990;50:490–3,HI

Atrioventricular
- Unusual opening of coronary sinus in atrioventricular septal defects (Wilcox, Anderson, & others) 1990;50:767–70*
- Valve insufficiency after atrioventricular septal defect repair: differences between patients with and without Down's syndrome? (Marino) 1990;50:854,CP; Reply (Abbruzzese, Napoleone, & others) 1990;50:854,CP

Autopsy
- Aortic dissection with the entrance tear in transverse aorta: analysis of 12 autopsy patients (Roberts & Roberts) 1990; 50:762–6*

Bacteria
- Bilateral pulmonary nodules caused by *Dirofilaria immitis* (Bradham, Locklair, & Grimball) 1990;50:312–3,CR

Biochemistry
- Preservation of myocardial function and biochemistry after blood and oxygenated crystalloid cardioplegia during cardiac arrest (Coetzee, Roussouw, & others) 1990;50:230–7*

Biopsy
- Chest wall recurrence of lung cancer after transthoracic fine needle aspiration biopsy (Hix) 1990;50:1020–1,CP

Björk-Shiley valve
- Development of an artificial heart valve (Björk) 1990;50:151–4,CL

Blood
- Acute preoperative plasmapheresis and established blood conservation techniques (Boldt, Kling, & others) 1990;50: 62–8*
- Aspirin, blood loss, and transfusion (Gay) 1990;50:345,ED
- Blood conservation in cardiac surgery (Scott, Kessler, & Wernly) 1990;50:843–51,CV
- Decreased vascular compliance after reimplantation of the left lower lobe in young pigs (Johnson, Teague, & others) 1990;50:277–80*
- How should the risk of bank blood transfusion in open heart operations be minimized? (Santoli, Scrofani, & Santoli) 1990;50:1021–2,CP
- Low-dose preoperative aspirin therapy, postoperative blood loss, and transfusion requirements (Taggart, Siddiqui, & Wheatley) 1990;50:425–8*
- Oxygenated cardioplegia: blood is a many splendored thing (Buckberg) 1990;50:175–7,ED
- Preservation of myocardial function and biochemistry after blood and oxygenated crystalloid cardioplegia during cardiac arrest (Coetzee, Roussouw, & others) 1990;50:230–7*
- Risks of blood exposure to the cardiac surgical team (Pate) 1990;50:248–50*

Blood flow
- Effects of left ventricular assist for cardiogenic shock on cardiac function and organ blood flow distribution (Sukehiro & Flameng) 1990;50:374–83*
- Flow capacity of the human retrograde internal mammary artery: surgical considerations (González-Santos, Bastida, & others) 1990;50:360–6*
- Inadequate flow of the internal mammary artery: another complication of sternal overretraction (Jebara, Acker, & others) 1990;50:336–7,CP

Blood pressure
- Flow capacity of the human retrograde internal mammary artery: surgical considerations (González-Santos, Bastida, & others) 1990;50:360–6*

Body fluids
- Risks of blood exposure to the cardiac surgical team (Pate) 1990;50:248–50*

Book reviews
- *Atlas of Cardiothoracic Surgery*, by Edmunds, Norwood, & Low (Tarazi) 1990;50:412,RV
- *General Thoracic Surgery, Third Edition*, edited by Shields (Keagy) 1990;50:68,RV

Book reviews (cont'd)
- *Interventional and Surgical Cardiovascular Pathology: Clinical Correlations and Basic Principles*, by Schoen (Orszulak) 1990;50:790,RV
- *Medical Management of the Cardiac Surgical Patient*, edited by Gray & Matloff (Jackson) 1990;50:819,RV
- *Preoperative Cardiac Assessment*, edited by Mangano (Swain) 1990;50:986,RV
- *Surgery of the Chest, 5th Edition, Volume II*, edited by Sabiston & Spencer (Dobell) 1990;50:825,RV
- *Surgery of the Thymus*, edited by Merlini, Clarke, & Dusmet (Mulder) 1990;50:1018,RV
- *Thoracic Trauma*, edited by Hood, Boyd, & Culliford (Mansour) 1990;50:1009,RV
- *Transplant—A Heart Surgeon's Account of the Life-and-Death Dramas of the New Medicine*, by First (Roe) 1990;50:203,RV
- *Vascular Surgery, Third Edition*, edited by Rutherford (Graham) 1990;50:135,RV

Boxing
- Pharyngoesophageal perforation after blunt neck trauma (Niezgoda, McMenamin, & Graeber) 1990;50:615–7*

Brachytherapy
- Neodymium:YAG laser and brachytherapy in the management of inoperable bronchogenic carcinoma (Miller & Phillips) 1990;50:190–6*

Bronchi
- Aortobronchial fistula after an aortic operation (Ishizaki, Tada, & others) 1990;50:975–7,CR
- Behavior of small bronchial carcinomas (Shields) 1990;50:691–2,ED
- Bronchial and thymic carcinoids and the ectopic ACTH syndrome (Odell) 1990;50:5–6,ED
- Bronchoesophageal fistula with an esophageal web (Turrentine, Kesler, & Mohamed) 1990;50:473–5,CR
- Esophagobronchial fistula and mediastinal tuberculosis (Raghu & Dillard) 1990;50:647–9,CR
- Intraoperative bronchial aspiration of ruptured pulmonary hydatid cysts (Saidi & Rezvan-Nobahar) 1990;50:631–6*
- Neodymium:YAG laser and brachytherapy in the management of inoperable bronchogenic carcinoma (Miller & Phillips) 1990;50:190–6*
- New approach to the surgical treatment of Swyer-James-MacLeod syndrome (Vishnevsky & Nikoladze) 1990;50:103–4*
- Results in 104 patients undergoing bronchoplastic procedures for bronchial lesions (Watanabe, Shimizu, & others) 1990;50:607–14*
- Surgical management of pulmonary metastatic leiomyosarcoma with gross endobronchial extension (Warren, Bleck, & others) 1990;50:739–42*
- Tracheal and main bronchial disruptions after blunt chest trauma: presentation and management (Baumgartner, Sheppard, & others) 1990;50:569–74*
- Traumatic bronchial rupture and other major thoracic injuries (Benfield) 1990;50:523,ED

Budd-Chiari syndrome
- Liver transplantation with atrioatrial anastomosis for Budd-Chiari syndrome (Carrel, Decurtins, & others) 1990;50:658–60,CR
- Membranotomy for Budd-Chiari syndrome (Victor, Jayanthi, & Madanagopalan) 1990;50:512–3,CP

Bypass
- Internal mammary artery bypass after the arterial switch operation (Rheuban, Kron, & Bulatovic) 1990;50:125–6,CR

Calcium
- Acute hypercalcemic crisis after an open heart operation (Siclari, Herrmann, & Dralle) 1990;50:831–2,CR
- The concentration of calcium in neonatal cardioplegia (Zweng, Iannettoni, & others) 1990;50:262–7*

Cannula
- Facile retrograde cardioplegia: transatrial cannulation of the coronary sinus (Gundry, Sequiera, & others) 1990;50:882–7*
- Improved cannulation method for extracorporeal membrane oxygenation (Read, St. Cyr, & others) 1990;50:670–1,HW
- Rapid placement of the Hemopump and hemofiltration cannula (Duncan, Burnett, & others) 1990;50:667–9,HW

Carbon dioxide
- Thoracoscopic treatment of spontaneous pneumothorax using carbon dioxide laser (Wakabayashi, Brenner, & others) 1990;50:786–90*

Carcinoid tumor
- Bronchial and thymic carcinoids and the ectopic ACTH syndrome (Odell) 1990;50:5–6,ED
- Carcinoid tumors of the thymus (Economopoulos, Lewis, & others) 1990;50:58–61*
- Management of the ectopic ACTH syndrome due to thoracic carcinoids (Pass, Doppman, & others) 1990;50:52–7*

Carcinoma. *See* Neoplasms.

Cardiomyopathy, hypertrophic
- Subvalvar aortic stenosis: timing of operation (Douville, Sade, & others) 1990;50:29–34*

Cardioplegia
- Cardioplegia for transplantation: failure of extracellular solution compared with Stanford or UW solution (Gott, Pan-Chih, & others) 1990;50:348–54*
- Cardioplegia-induced damage to ischemic immature myocardium is independent of oxygen availability (Baker, Boerboom, & Olinger) 1990;50:934–9*
- The concentration of calcium in neonatal cardioplegia (Zweng, Iannettoni, & others) 1990;50:262–7*
- Easy cardioplegia delivery, aortic venting, and reperfusion technique (Kauten) 1990;50:833–5,HW
- Endothelial cell toxicity of solid-organ preservation solutions (von Oppell, Pfeiffer, & others) 1990;50:902–10*
- Facile retrograde cardioplegia: transatrial cannulation of the coronary sinus (Gundry, Sequiera, & others) 1990;50:882–7*
- Myocardial preservation using lidocaine blood cardioplegia (Fiore, Naunheim, & others) 1990;50:771–5*
- Myocardial protection by retrograde cardioplegia in arterial switch operation (Yonenaga, Yasui, & others) 1990;50:238–42*
- Oxygenated cardioplegia: blood is a many splendored thing (Buckberg) 1990;50:175–7,ED
- Preservation of myocardial function and biochemistry after blood and oxygenated crystalloid cardioplegia during cardiac arrest (Coetzee, Roussouw, & others) 1990;50:230–7*

Cardiopulmonary bypass
- Antimicrobial prophylaxis for open heart operations (Miedzinski, Callaghan, & others) 1990;50:800–7*
- Beware centrifugal pumps: not a one-way street, but a potentially dangerous "siphon" (Kolff, McClurken, & Alpern) 1990;50:512,CP
- Blood conservation in cardiac surgery (Scott, Kessler, & Wernly) 1990;50:843–51,CV
- Choreoathetosis after deep hypothermia without circulatory arrest (DeLeon, Ilbawi, & others) 1990;50:714–9*

Clinical experience with portable cardiopulmonary bypass in cardiac arrest patients (Hartz, LoCicero, & others) 1990;50: 437–41*
Complement activation and lung permeability during cardiopulmonary bypass (Tennenberg, Clardy, & others) 1990; 50:597–601*
Cor triatriatum: clinical presentation and surgical results in 12 patients (Rodefeld, Brown, & others) 1990;50:562–8*
Easy cardioplegia delivery, aortic venting, and reperfusion technique (Kauten) 1990;50:833–5,HW
Hypoxemia during cardiopulmonary bypass (Belcher & Jones) 1990;50:336,CP
Liver transplantation with atrioatrial anastomosis for Budd-Chiari syndrome (Carrel, Decurtins, & others) 1990;50: 658–60,CR
Myocardial protection by retrograde cardioplegia in arterial switch operation (Yonenaga, Yasui, & others) 1990;50:238–42*
Oxygenator failure (Crowley & Brown) 1990;50:859–60,CP
Reversal of protamine-induced catastrophic pulmonary vasoconstriction by prostaglandin E_1 (Whitman, Martel, & others) 1990;50:303–5,CR
Role of perfusion pressure and flow in major organ dysfunction after cardiopulmonary bypass (Slogoff, Reul, & others) 1990;50:911–8*
Transatrial approach revisited (Khonsari & Sintek) 1990;50: 1002–3,HW
Ventricular perforation with valvoplasty (Aaron & Alyono) 1990;50:1002,CP; Reply (Robertson) 1990;50:1022–3,CP

Case reports
Acute hypercalcemic crisis after an open heart operation (Siclari, Herrmann, & Dralle) 1990;50:831–2,CR
Acute symptoms due to a huge duplication cyst ruptured into the esophagus (Nakahara, Fujii, & others) 1990;50:309–11,CR
Aortobronchial fistula after an aortic operation (Ishizaki, Tada, & others) 1990;50:975–7,CR
Bilateral pulmonary nodules caused by *Dirofilaria immitis* (Bradham, Locklair, & Grimball) 1990;50:312–3,CR
Bronchial anomaly of the right upper lobe (Read, St. Cyr, & others) 1990;50:980–1,CR
Bronchoesophageal fistula with an esophageal web (Turrentine, Kesler, & Mohamed) 1990;50:473–5,CR
Catastrophic thrombosis of porcine aortic bioprostheses (Baciewicz, del Rio, & others) 1990;50:817–9,CR
Circumflex artery ventricular fistula and pseudoaneurysm after mitral reoperation (Sutter, Goldman, & others) 1990;50: 826–7,CR
Coexisting thymic carcinoid tumor and thymoma (Mizuno, Masaoka, & others) 1990;50:650–2,CR
Combined repair of transposed great arteries and total anomalous pulmonary venous connection (Amodeo, Corno, & others) 1990;50:820–1,CR
Complete foregut duplication (Dresler, Patterson, & others) 1990;50:306–8,CR
Congenital tracheoesophageal fistula associated with carcinoma of the lung in an adult (Campbell & Salisbury) 1990;50:978–9,CR
Coronary revascularization in a child with Kawasaki disease: use of right gastroepiploic artery (Takeuchi, Gomi, & others) 1990;50:294–6,CR
Cystic medionecrosis of the coronary arteries and fatal coronary vasospasm (Segal, Ratliff, & Cosgrove) 1990;50:653–5,CR
Disruption of a modified Blalock-Taussig shunt by rapid deceleration injury (Joyce & McGrath) 1990;50:124,CR
Esophageal perforation in a patient with acquired immunodeficiency syndrome (Adkins, Raccuia, & Acinapura) 1990; 50:299–300,CR
Esophagobronchial fistula and mediastinal tuberculosis (Raghu & Dillard) 1990;50:647–9,CR
Extended en bloc resection of a primary mediastinal parathyroid carcinoma (Putnam, Schantz, & others) 1990;50:138–40,CR
Fatal ectopic thymoma (Jansen & Johnson) 1990;50:469–70,CR
Internal mammary artery bypass after the arterial switch operation (Rheuban, Kron, & Bulatovic) 1990;50:125–6,CR
Internal mammary artery graft to pulmonary vasculature fistula: a cause of recurrent angina (Johnson, Schmaltz, & others) 1990;50:297–8,CR
Intramural ventricular fibroma in infancy: survival after partial excision in 2 patients (Ceithaml, Midgley, & others) 1990; 50:471–2,CR
Isolated left carotid artery in CHARGE association: diagnosis and repair (Ghalili, Issenberg, & others) 1990;50:130–2,CR
Job's syndrome: a rare cause of recurrent lung abscess in childhood (Lui & Inculet) 1990;50:992–4,CR
Left recurrent laryngeal nerve paralysis: an unusual presentation of histoplasmosis (Gilbert, Murray, & others) 1990;50: 987–8,CR
Left superior vena cava: a pitfall in computed tomographic diagnosis with surgical implications (Wong & Goldstraw) 1990;50:656–7,CR
Liver transplantation with atrioatrial anastomosis for Budd-Chiari syndrome (Carrel, Decurtins, & others) 1990;50: 658–61,CR
Malignant fibrous histiocytoma of the trachea (Randleman, Unger, & Mansour) 1990;50:458–9,CR
Malignant thymoma presenting as intracardiac tumor and superior vena caval obstruction (Airan, Sharma, & others) 1990;50:989–91,CR
Management of aortobronchial fistula with graft replacement and omentopexy (Paull & Keagy) 1990;50:972–4,CR
Management of mycotic rupture of the ascending aorta after heart-lung transplantation (Albes, Haverich, & others) 1990;50:982–3,CR
Mediastinal hibernoma, a rare tumor (Ahn & Harvey) 1990;50: 828–30,CR
Norwood operation for univentricular heart with subaortic stenosis in the neonate (Tchervenkov, Béland, & others) 1990;50:822–5,CR
Polytetrafluoroethylene graft for spontaneous coronary dissection: 7-year follow-up (McCullough, Abel, & Rubenstein) 1990;50:663–4,CR
Presumed Kawasaki disease resulting in multiple coronary artery aneurysms in an adult (Brevard & Smith) 1990;50: 291–3,CR
Primary mediastinal leiomyoma (Shaffer, Pugatch, & Sugarbaker) 1990;50:301–2,CR
Resection of the superior vena cava for primary lung cancer: 5 years' survival (Inoue, Shohtsu, & others) 1990;50:661–2,CR
Retrotracheal goiter: a diagnostic and therapeutic problem (Waldron, Coffey, & others) 1990;50:33–5,CR
Reversal of protamine-induced catastrophic pulmonary vasoconstriction by prostaglandin E_1 (Whitman, Martel, & others) 1990;50:303–5,CR
Right atrial perforation by a temporary epicardial pacing wire (Smith & Tatoulis) 1990;50:141–2,CR

Case reports (cont'd)
- Right pneumonectomy syndrome in infancy treated with an expandable prosthesis (Rasch, Grover, & others) 1990; 50:127–9,CR
- Scimitar syndrome: cine magnetic resonance imaging demonstration of anomalous pulmonary venous drainage (Baxter, McFadden, & others) 1990;50:121–3,CR
- Severe hemolysis after incomplete mitral valve repair (Wilson, Rath, & others) 1990;50:136–7,CR
- Silastic prothesis plombage for right postpneumonectomy syndrome (Riveron, Adams, & others) 1990;50:465–6,CR
- Squamous carcinoma metastatic to the sternum (D'Orazio, Naunheim, & others) 1990;50:984–6,CR
- Steal phenomenon caused by a parallel branch of the internal mammary artery (Schmid, Heublein, & others) 1990;50: 463–4,CR
- Successful Fontan-type operation for a nonresectable right ventricular tumor (Dietl) 1990;50:814–6,CR
- Successful surgical treatment of incessant automatic atrial tachycardia with atrial aneurysm (Miyamura, Nakagomi, & others) 1990;50:476–8,CR
- Swan-Ganz catheter-induced massive hemoptysis and pulmonary artery false aneurysm (Feng, Singh, & others) 1990; 50:644–6,CR
- Systemic venous aneurysms (Yokomise, Nakayama, & others) 1990;50:460–2,CR
- Tension pneumopericardium as a complication of single-lung transplantation (Cochrane, Mitchell, & others) 1990;50: 808–10,CR
- Tubular supravalvar aortic stenosis: replacement of ascending aorta in a young patient (Gargiulo, Zannini, & others) 1990;50:467–8,CR
- Vagal schwannoma (Heitmiller, Labs, & Lipsett) 1990;50:811–3,CR

Catheterization
- Infant ECMO cannulation technique allowing preservation of carotid and jugular vessels (Karl, Iyer, & others) 1990;50: 488–9,HW
- Rapid placement of the Hemopump and hemofiltration cannula (Duncan, Burnett, & others) 1990;50:667–9,HW
- Swan-Ganz catheter-induced massive hemoptysis and pulmonary artery false aneurysm (Feng, Singh, & others) 1990; 50:644–6,CR

Cellulose
- Paraplegia associated with the use of oxidized cellulose in posterolateral thoracotomy incisions (Short) 1990;50:288–90*

Child
- Coronary revascularization in a child with Kawasaki disease: use of right gastroepiploic artery (Takeuchi, Gomi, & others) 1990;50:294–6,CR
- Critical aortic stenosis in the first month of life: surgical results in 26 infants (Karl, Sano, & others) 1990;50:105–9*
- Disruption of a modified Blalock-Taussig shunt by rapid deceleration injury (Joyce & McGrath) 1990;50:124,CR
- Feasibility of reversible pulmonary artery banding: early results and intermediate-term follow-up (Epstein, Duncan, & others) 1990;50:94–7*
- Job's syndrome: a rare cause of recurrent lung abscess in childhood (Lui & Inculet) 1990;50:992–4,CR
- Late results of aortic valvotomy for congenital valvar aortic stenosis (DeBoer, Robbins, & others) 1990;50:69–73*
- Long-term results of total repair of tetralogy of Fallot in childhood (Horneffer, Zahka, & others) 1990;50:179–85*
- Pediatric heart transplantation (Smyth, Scott, & others) 1990; 50:333,CP; Reply (Trento) 1990;50:333,CP
- Prolonged extracorporeal life support of pediatric and adolescent cardiac transplant patients (Delius, Zwischenberger, & others) 1990;50:791–5*
- Relief of tracheal compression by aortopexy (Clevenger, Othersen, & Smith) 1990;50:524–9*
- Repair of complete atrioventricular septal defect with tetralogy of Fallot (Ilbawi, Cua, & others) 1990;50:407–12*
- Successful surgical treatment of incessant automatic atrial tachycardia with atrial aneurysm (Miyamura, Nakagomi, & others) 1990;50:476–8,CR
- Tubular supravalvar aortic stenosis: replacement of ascending aorta in a young patient (Gargiulo, Zannini, & others) 1990;50:467–8,CR
- Value of transesophageal echocardiography during repair of congenital heart defects (Dan, Bonato, & others) 1990;50: 637–43*

Chordae tendineae
- Repair of flail anterior leaflets of tricuspid and mitral valves by cusp remodeling (Sutlic, Schmid, & Borst) 1990;50:927–30*

Choreoathetosis
- Choreoathetosis after deep hypothermia without circulatory arrest (DeLeon, Ilbawi, & others) 1990;50:714–9*

Circulation
- New technique for the arterial switch operation in difficult situations (Takeuchi & Katogi) 1990;50:1000–1,HW
- Role of perfusion pressure and flow in major organ dysfunction after cardiopulmonary bypass (Slogoff, Reul, & others) 1990;50:911–8*

Classics in thoracic surgery
- Collis gastroplasty: origin and evolution (Adler) 1990;50:839–42,CL
- Development of an artificial heart valve (Björk) 1990;50:151–4,CL
- Origins of intraaortic balloon pumping (Kantrowitz) 1990;50: 672–4,CL
- Postinfarction ventricular septal defect repair: retrospective thoughts and historical perspectives (Daggett) 1990;50: 1006–9,CL
- Use of the allograft aortic valved conduit (Campbell & Clarke) 1990;50:320–2,CL

Codes
- Using ICD-9-CM and CPT in the Nineties (Levitsky) 1990;50: 519–20,ED

Cold
- Improved myocardial preservation during cold storage using substrate enhancement (Haan, Lazar, & others) 1990;50: 80–5*

Collective reviews
- Intrathoracic tumors of the vagus nerve (Dabir, Piccione, & Kittle) 1990;50:494–7,CT
- Mid-descending aortic traumatic aneurysms (Rabinsky, Sidhu, & Wagner) 1990;50:155–60,CT

Complement
- Complement activation with bubble and membrane oxygenators in aortocoronary bypass grafting (Videm, Fosse, & others) 1990;50:387–91*
- Complement activation and lung permeability during cardiopulmonary bypass (Tennenberg, Clardy, & others) 1990; 50:597–601*

Computed tomography. *See* Tomography, computed.

Congenital defects. *See* Abnormalities

Contamination
Air contamination in open heart surgery with disposable coveralls, gowns, and drapes (Verkkala, Mäkelä, & others) 1990;50:757–61*
Contractility
Timing of operation for aortic regurgitation: relation to postoperative contractile state (Taniguchi, Nakano, & others) 1990;50:779–85*
Cor triatriatum
Cor triatriatum: clinical presentation and surgical results in 12 patients (Rodefeld, Brown, & others) 1990;50:562–8*
Coronary
Facile retrograde cardioplegia: transatrial cannulation of the coronary sinus (Gundry, Sequiera, & others) 1990;50:882–7*
Unusual opening of coronary sinus in atrioventricular septal defects (Wilcox, Anderson, & others) 1990;50:767–70*
Coronary artery bypass
Arteriosclerosis of the gastroepiploic and internal thoracic arteries (Suma & Takanashi) 1990;50:413–6*
Combined internal mammary artery graft for coronary artery revascularization (Bakay, Akçevin, & others) 1990;50:553–6*
Complement activation with bubble and membrane oxygenators in aortocoronary bypass grafting (Videm, Fosse, & others) 1990;50:387–91*
Coronary artery bypass grafting without splenectomy (Bowman) 1990;50:330–1,CP; Reply (Koike) 1990;50:331,CP
Determinants of pulmonary function in patients undergoing coronary bypass operations (Shapira, Zabatino, & others) 1990;50:268–73*
Five-year results of coronary bypass grafting for patients older than 70 years: role of internal mammary artery (Azariades, Fessler, & others) 1990;50:940–5*
Free gastroepiploic artery graft (Mills & Everson) 1990;50:858,CP
Guidelines for transfusion support in patients undergoing coronary artery bypass grafting (Goodnough, Johnston, & others) 1990;50:675–83,CV
Pleuropulmonary morbidity: internal thoracic artery versus saphenous vein graft (Hurlbut, Myers, & others) 1990;50:959–64*
Right atrial perforation by a temporary epicardial pacing wire (Smith & Tatoulis) 1990;50:141–2,CR
Risks of blood exposure to the cardiac surgical team (Pate) 1990;50:248–50*
Spasm of arterial conduits in aorta-coronary bypass grafting (van Son & Vincent) 1990;50:1019,CP; Reply (Suma) 1990;50:1019–20,CP
Squamous carcinoma metastatic to the sternum (D'Orazio, Naunheim, & others) 1990;50:984–6,CR
Surgical standby for percutaneous transluminal coronary angioplasty: a survey of patterns of practice (Cameron, Stinson, & others) 1990;50:35–9*
Coronary disease
Cardiac transplantation (Griffith) 1990;50:161–2,KR
Cystic medionecrosis of the coronary arteries and fatal coronary vasospasm (Segal, Ratliff, & Cosgrove) 1990;50:653–5,CR
Intraoperative ultrasonic imaging of the ascending aorta in ischemic heart disease (Ohtaki, Itoh, & others) 1990;50:539–42*
Correspondence
Anomalous left coronary artery surgery (Alexi-Meskhishvili) 1990;50:511,CP; Reply (Clarke) 1990;50:512,CP
Beware centrifugal pumps: not a one-way street, but a potentially dangerous "siphon" (Kolff, McClurken, & Alpern) 1990;50:512,CP
Cardiopulmonary cystosarcoma (Shuman) 1990;50:510,CP; Reply (Tyers) 1990;50:510–11,CP
Certification of perfusionists (Fosburg, Anderson, & others) 1990;50:508,CP
Chest wall recurrence of lung cancer after transthoracic fine needle aspiration biopsy (Hix) 1990;50:1020–1,CP
Choice of surgical clips for mammary artery take-down (Hovaguimian & Floten) 1990;50:856,CP; Reply (Gold) 1990;50:856–7,CP
Closed chest cardiac massage (Zuhdi) 1990;50:331–2,CP; (Robicsek) 1990;50:332–3,CP
Computed tomography in selecting patients for mediastinoscopy (Van Schil) 1990;50:163,CP; Reply (Thermann) 1990;50:163,CP
Coronary artery bypass grafting without splenectomy (Bowman) 1990;50:330–1,CP; Reply (Koike) 1990;50:331,CP
Descending necrotizing mediastinitis (Horowitz, Sosa, & Lickstein) 1990;50:859–60,CP
Diaphragmatic plication (Ribet) 1990;50:855–6,CP
Double-barrel suction device (Robicsek) 1990;50:509,CP
Efficacy and benefit of mediastinal computed tomography (Gaer & Goldstraw) 1990;50:508,CP
Efficacy of pulmonary artery versus left ventricular venting (Garibaldi) 1990;50:685,CP; Reply (Mitchell) 1990;50:686,CP
Elevated hemidiaphragm after cardiac operations (Esposito) 1990;50:166–7,CP; Reply (Curtis) 1990;50:167,CP
Endoscopic relief of airway obstruction (Cavaliere & Ardigo) 1990;50:163,CP; Reply (Mathisen & Grillo) 1990;50:164,CP
Esophagectomy for Barrett's esophagus (Weil) 1990;50:858–9,CP; Reply (Altorki) 1990;50:859,CP
Free gastroepiploic artery graft (Mills & Everson) 1990;50:858,CP
How should the risk of bank blood transfusion in open heart operations be minimized? (Santoli, Scrofani, & Santoli) 1990;50:1021–2,CP
Hypoxemia during cardiopulmonary bypass (Belcher & Jones) 1990;50:336,CP
Inadequate flow of the internal mammary artery: another complication of sternal overretraction (Jebara, Acker, & others) 1990;50:336–7,CP
Individual ligation technique (Meyer) 1990;50:1023,CP
Infection and Cell-Saver use (Nessly) 1990;50:509–10,CP
Internal mammary artery marker (Kyger) 1990;50:333–4,CP
Internal mammary artery-to-pulmonary artery shunt in infants (Sakakibara, Hori & others) 1990;50:857–8,CP
Intraoperative photodynamic therapy for malignant mesothelioma (Pass, Tochner, & others) 1990;50:687–8,CP
Intraoperative transesophageal echocardiography for hypertrophic cardiomyopathy (Eng, Nair, & others) 1990;50:513–4,CP
Is systemic cooling essential for open heart surgery? (Victor & Kabeer) 1990;50:334–5,CP
Left ventricular outflow tract obstruction with aortic arch anomalies and ventricular septal defect (Sell) 1990;50:854–5,CP; Reply (Iwahara, Ino, Yabuta) 1990;50:855,CP
Malignant fibrous histiocytoma (Laas & Schmid) 1990;50:164,CP; Reply (Lazar) 1990;50:165,CP; (Craddock) 1990;50:165,CP
Management of Jehovah's Witness patients (Kyger & Blakestad) 1990;50:167,CP

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Membranotomy for Budd-Chiari syndrome (Victor, Jayanthi, & Madanagopalan) 1990;50:512–3,CP
Organ procurement for pulmonary transplantation (Ribet & Pruvot) 1990;50:335,CP; Reply (Griffith) 1990;50:335–6,CP
Oxygenator failure (Crowley & Brown) 1990;50:859–60,CP
Pediatric heart transplantation (Smyth, Scott, & others) 1990;50:333,CP; Reply (Trento) 1990;50:333,CP
Plastic struts for delayed sternal closure (Majid) 1990;50:1021,CP
Polytetrafluoroethylene posterior annuloplasty for mitral regurgitation (Fundarò, Salati, & others) 1990;50:165–6,CP; Reply (Cosgrove) 1990;50:166,CP
Postirradiation pericardiectomy (Eckstein) 1990;50:685,CP
Postoperative drainage and pericardial effusion (Bryan & Angelini) 1990;50:330,CP; Reply (Smulders, Moulijn, & Koolen) 1990;50:330,CP
Repair of thoracic dissections (Hammond) 1990;50:330,CP
Respiratory function after thoracic operations (Hallfeldt, Knoefel, & others) 1990;50:688,CP
Retrotracheal mediastinal goiter with contralateral extension (Ribet) 1990;50:684,CP; Reply (Van Schil) 1990;50:684,CP
Spasm of arterial conduits in aorta-coronary bypass grafting (van Son & Vincent) 1990;50:1019,CP; Reply (Suma) 1990;50:1019–21,CP
Sternotomy infections in infants (Stahl & Kopf) 1990;50:337,CP; Reply (Terranova & Crawford) 1990;50:337,CP
Thymectomy for myasthenia gravis (Jaretski) 1990;50:686,CP; Reply (Mulder) 1990;50:687,CP
Valve insufficiency after atrioventricular septal defect repair: differences between patients with and without Down's syndrome? (Marino) 1990;50:854,CP; Reply (Abbruzzese, Napoleone, & others) 1990;50:854,CP
Vein graft permeability (Kennedy, Tedgui, & Piwnica) 1990;50:334,CP; Reply (Finck, Mashburn, & others) 1990;50:334,CP
Ventricular perforation with valvoplasty (Aaron & Alyono) 1990;50:1022,CP; Reply (Robertson) 1990;50:1022–3,CP

Creatine kinase
Selective monitoring of patients with suspected blunt cardiac injury (Reif, Justice, & others) 1990;50:530–3*

Current reviews
Blood conservation in cardiac surgery (Scott, Kessler, & Wernly) 1990;50:843–51,CV
Guidelines for transfusion support in patients undergoing coronary artery bypass grafting (Goodnough, Johnston, & others) 1990;50:675–83,CV
Intraoperative echocardiography (Lazar & Plehn) 1990;50:1010–8,CV
Paradoxical hypertension after repair of coarctation of the aorta: a review of its causes (Sealy) 1990;50:323–9,CV

Cyst
Acute symptoms due to a huge duplication cyst ruptured into the esophagus (Nakahara, Fujii, & others) 1990;50:309–11,CR
Intraoperative bronchial aspiration of ruptured pulmonary hydatid cysts (Saidi & Rezvan-Nobahar) 1990;50:631–6*

Debridement
Ultrasonic debridement during mitral valve reconstruction for calcified mitral stenosis (Nakano, Mitsuno, & others) 1990;50:923–6*

Decalcification
Aortic valve repair for aortic stenosis in adults (Shapira, Lemole, & others) 1990;50:110–20*

Dialysis
Internal mammary artery revascularization in the patient on long-term renal dialysis (Blakeman, Sullivan, & others) 1990;50:776–8*

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Diaphragmatic plication (Ribet) 1990;50:855–6,CP
Elevated hemidiaphragm after cardiac operations (Esposito) 1990;50:166–7,CP; Reply (Curtis) 1990;50:167,CP

Digoxin
Prophylactic digitalization for thoracotomy: a reassessment (Ritchie, Bowe, & Gibbons) 1990;50:86–8*

Disposable equipment
Air contamination in open heart surgery with disposable coveralls, gowns, and drapes (Verkkala, Mäkelä, & others) 1990;50:757–61*

Dissection
Aortic dissection with the entrance tear in transverse aorta: analysis of 12 autopsy patients (Roberts & Roberts) 1990;50:762–6*
Easy dissection of hard and thickened pericardium on constrictive pericarditis (Oku, Sawada, & others) 1990;50:998–9,HW
Surgical epicardial ablation of left ventricular pathway using sling exposure (Guiraudon, Klein, & others) 1990;50:968–71*

Diuresis
Diuresis in hemodynamically compromised patients: continuous furosemide infusion (Magovern & Magovern) 1990;50:482–4,HW

Dog
Artificial mitral valve chordae: experimental and clinical experience (Zussa, Frater, & others) 1990;50:367–73*
Cardioplegia for transplantation: failure of extracellular solution compared with Stanford or UW solution (Gott, Pan-Chih, & others) 1990;50:348–54*
Feasibility of reversible pulmonary artery banding: early results and intermediate-term follow-up (Epstein, Duncan, & others) 1990;50:94–7*
Leukocyte depletion results in excellent heart-lung function after 12 hours of storage (Pillai, Bando, & others) 1990;50:211–4*
Modification of supraventricular tachyarrhythmias by stimulating atrial neurons (Ali, Butler, & others) 1990;50:251–6*
Oxygen free radical scavengers decrease reperfusion injury in lung transplantation (Detterbeck, Keagy, & others) 1990;50:204–10*
Tracheal reconstruction with polytetrafluoroethylene graft in dogs (Cull, Lally, & others) 1990;50:899–901*

Drainage
Descending necrotizing mediastinitis (Horowitz, Sosa, & Lickstein) 1990;50:859–60,CP

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Intraoperative bronchial aspiration of ruptured pulmonary hydatid cysts (Saidi & Rezvan-Nobahar) 1990;50:631–6*

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Intraoperative echocardiography (Lazar & Plehn) 1990;50:1010–8,CV
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Pericardial window: mechanisms of efficacy (Sugimoto, Little, & others) 1990;50:442–5*
Selective monitoring of patients with suspected blunt cardiac injury (Reif, Justice, & others) 1990;50:530–3*

Subaortic obstruction: intraoperative echocardiography as an adjunct to operation (Sreeram, Sutherland, & others) 1990; 50:579–85*
Surgical treatment of cardiac myxomas: long-term results (Sellke, Lemmer, & others) 1990;50:557–61*
Value of transesophageal echocardiography during repair of congenital heart defects (Dan, Bonato, & others) 1990;50: 637–43*

Economics
Extension of donor criteria in cardiac transplantation: surgical risk versus supply-side economics (Sweeney, Lammermeier, & others) 1990;50:7–11*

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Paraplegia associated with thoracotomy (Walker) 1990;50: 178,ED
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Effusion
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Systemic arterial air embolism in penetrating lung injury (Estrera, Pass, & Platt) 1990;50:257–61*

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Thoracoplasty: current application to the infected pleural space (Horrigan & Snow) 1990;50:695–9*
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Clinical experience with the Medtronic-Hall valve prosthesis (Keenan, Armitage, & others) 1990;50:748–53*

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Endothelium
Endothelial cell toxicity of solid-organ preservation solutions (von Oppell, Pfeiffer, & others) 1990;50:902–10*

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Surgical epicardial ablation of left ventricular pathway using sling exposure (Guiraudon, Klein, & others) 1990;50:968–71*

Epidemiology
Coarctation of the aorta (van Son) 1990;50:852–3,KR
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Esophagus
Acquired benign esophagorespiratory fistula: report of 16 consecutive cases (Gerzić, Rakić, & Randjelović) 1990;50: 724–7*
Acute symptoms due to a huge duplication cyst ruptured into the esophagus (Nakahara, Fujii, & others) 1990;50:309–11,CR
Bronchoesophageal fistula with an esophageal web (Turrentine, Kesler, & Mohamed) 1990;50:473–5,CR
Congenital tracheoesophageal fistula associated with carcinoma of the lung in an adult (Campbell & Salisbury) 1990;50:978–9,CR
Esophageal perforation in a patient with acquired immunodeficiency syndrome (Adkins, Raccuia, & Acinapura) 1990; 50:299–300,CR
Esophageal perforation: a therapeutic challenge (Attar, Hankins, & others) 1990;50:45–51*
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Platysma musculocutaneous flap for reconstruction of trachea in esophageal cancer (Sodeyama, Matsuo, & others) 1990; 50: 485–7,HW

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Cardioplegia for transplantation: failure of extracellular solution compared with Stanford or UW solution (Gott, Pan-Chih, & others) 1990;50:348–54*

Extracorporeal
Prolonged extracorporeal life support of pediatric and adolescent cardiac transplant patients (Delius, Zwischenberger, & others) 1990;50:791–5*

Extracorporeal membrane oxygenation
Infant ECMO cannulation technique allowing preservation of carotid and jugular vessels (Karl, Iyer, & others) 1990;50: 488–9,HW

Extracorporeal membrane oxygenation (cont'd)
  Prognostic determinants in extracorporeal membrane oxygenation for respiratory failure in newborns (Weber, Connors, & others) 1990;50:720–3*

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  Four years' experience with fibrin sealant in thoracic and cardiovascular surgery (Matthew, Spotnitz, & others) 1990;50:40–4*
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  Variations within the fibrous skeleton and ventricular outflow tracts in tetralogy of Fallot (Howell, Ho, & others) 1990;50:450–7*
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  Acquired benign esophagorespiratory fistula: report of 16 consecutive cases (Gerzić, Rakić, & Randjelović) 1990;50:724–7*
  Aortobronchial fistula after an aortic operation (Ishizaki, Tada, & others) 1990;50:975–7,CR
  Bronchoesophageal fistula with an esophageal web (Turrentine, Kesler, & Mohamed) 1990;50:473–5,CR
  Circumflex artery ventricular fistula and pseudoaneurysm after mitral reoperation (Sutter, Goldman, & others) 1990;50:826–7,CR
  Congenital tracheoesophageal fistula associated with carcinoma of the lung in an adult (Campbell & Salisbury) 1990;50:978–9,CR
  Esophagobronchial fistula and mediastinal tuberculosis (Raghu & Dillard) 1990;50:647–9,CR
  Internal mammary artery graft to pulmonary vasculature fistula: a cause of recurrent angina (Johnson, Schmaltz, & others) 1990;50:297–8,CR
  Management of aortobronchial fistula with graft replacement and omentopexy (Paull & Keagy) 1990;50:972–4,CR
Foreign body
  Giant chest wall tumor resulting from tissue reaction to foreign bodies (Fukai, Sano, & others) 1990;50:754–6*
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  Thoracic spinal fractures and aortic rupture: a significant and fatal association (Sturm, Hines, & Perry) 1990;50:931–3*
Furosemide
  Diuresis in hemodynamically compromised patients: continuous furosemide infusion (Magovern & Magovern) 1990;50:482–4,HW

Gastrointestinal tract
  Complete foregut duplication (Dresler, Patterson, & others) 1990;50:306–8,CR
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  Collis gastroplasty: origin and evolution (Adler) 1990;50:839–42,CL
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  Use of glue without graft replacement for type A dissections: a new surgical technique (Fabiani, Jebara, & others) 1990;50:143–5,HW
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  Retrotracheal goiter: a diagnostic and therapeutic problem (Waldron, Coffey, & others) 1990;50:133–5,CR
  Retrotracheal mediastinal goiter with contralateral extension (Ribet) 1990;50:684,CP; Reply (Van Schil) 1990;50:684,CP
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  Coronary artery bypass grafting without splenectomy (Bowman) 1990;50:330–1,CP; Reply (Koike) 1990;50:331,CP
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See also Transplantation.

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Leukocyte depletion results in excellent heart-lung function after 12 hours of storage (Pillai, Bando, & others) 1990;50:211–4*
Lidocaine
Myocardial preservation using lidocaine blood cardioplegia (Fiore, Naunheim, & others) 1990;50:771–5*
Life support care
Prolonged extracorporeal life support of pediatric and adolescent cardiac transplant patients (Delius, Zwischenberger, & others) 1990;50:791–5*
Ligation
Individual ligation technique (Meyer) 1990;50:1023,CP
Liver
Liver transplantation with atrioatrial anastomosis for Budd-Chiari syndrome (Carrel, Decurtins, & others) 1990;50:658–60,CR
Lung
Decreased vascular compliance after reimplantation of the left lower lobe in young pigs (Johnson, Teague, & others) 1990;50:277–80*
Determinants of pulmonary function in patients undergoing coronary bypass operations (Shapira, Zabatino, & others) 1990;50:268–73*
Individual ligation technique (Meyer) 1990;50:1023,CP
Systemic arterial air embolism in penetrating lung injury (Estrera, Pass, & Platt) 1990;50:257–61*
Lung disease
Job's syndrome: a rare cause of recurrent lung abscess in childhood (Lui & Inculet) 1990;50:992–4,CR
Long-term results of operation for non–small cell lung cancer in the elderly (Ishida, Yokoyama, & others) 1990;50:919–22*
Strategy for lymphadenectomy in lung cancer three centimeters or less in diameter (Ishida, Yano, & others) 1990;50:708–13*
Surgical management of pulmonary metastatic leiomyosarcoma with gross endobronchial extension (Warren, Bleck, & others) 1990;50:739–42*
Lung neoplasm
Chest wall recurrence of lung cancer after transthoracic fine needle aspiration biopsy (Hix) 1990;50:1020–1,CP
Congenital tracheoesophageal fistula associated with carcinoma of the lung in an adult (Campbell & Salisbury) 1990;50:978–9,CR
Resection of the superior vena cava for primary lung cancer: 5 years' survival (Inoue, Shohtsu, & others) 1990;50:661–2,CR
Role of postoperative radiation therapy in stage IIIa non–small cell lung cancer (Astudillo & Conill) 1990;50:618–23*
Lung transplantation
Organ procurement for pulmonary transplantation (Ribet & Pruvot) 1990;50:335,CP; Reply (Griffith) 1990;50:335–6,CP
Oxygen free radical scavengers decrease reperfusion injury in lung transplantation (Detterbeck, Keagy, & others) 1990;50:204–10*
Tension pneumopericardium as a complication of single-lung transplantation (Cochrane, Mitchell, & others) 1990;50:808–10,CR
Lymphadenopathy
Strategy for lymphadenectomy in lung cancer three centimeters or less in diameter (Ishida, Yano, & others) 1990;50:708–13*

Magnetic resonance imaging
Choice of surgical clips for mammary artery take-down (Hovaguimian & Floten) 1990;50:856,CP; Reply (Gold) 1990;50:856–7,CP
Diagnostic imaging and surgical treatment of dumbbell tumors of the mediastinum (Ricci, Rendina, & others) 1990;50:586–9*
Scimitar syndrome: cine magnetic resonance imaging demonstration of anomalous pulmonary venous drainage (Baxter, McFadden, & others) 1990;50:121–3,CR
Mammary artery
Combined internal mammary artery graft for coronary artery revascularization (Bakay, Akçevin, & others) 1990;50:553–6*
A fiber-optic retractor for harvesting the internal mammary artery (Angelini & Azzu) 1990;50:314–5,HW
Five-year results of coronary bypass grafting for patients older than 70 years: role of internal mammary artery (Azariades, Fessler, & others) 1990;50:940–5*
Flow capacity of the human retrograde internal mammary artery: surgical considerations (González-Santos, Bastida, & others) 1990;50:360–6*
Hemostatic technique for internal mammary artery anastomotic bleeding (Bhattacharyya, Trivedi, & others) 1990;50:316–7,HW

Mammary artery (cont'd)
Inadequate flow of the internal mammary artery: another complication of sternal overretraction (Jebara, Acker, & others) 1990;50:336–7,CP
Internal mammary artery bypass after the arterial switch operation (Rheuban, Kron, & Bulatovic) 1990;50:125–6*
Internal mammary artery graft to pulmonary vasculature fistula: a cause of recurrent angina (Johnson, Schmaltz, & others) 1990;50:297–8,CR
Internal mammary artery marker (Kyger) 1990;50:333–4,CP
Internal mammary artery revascularization in the patient on long-term renal dialysis (Blakeman, Sullivan, & others) 1990;50:776–8*
Pharmacological response of internal mammary artery and gastroepiploic artery (Koike, Suma, & others) 1990;50:384–6*
Selective and adjustable pericardial flap to protect internal mammary artery grafts (González-Santos, Bastida, & others) 1990;50:995–7,HW
Steal phenomenon caused by a parallel branch of the internal mammary artery (Schmid, Heublein, & others) 1990;50:463–4,CR

Mediastinitis
Descending necrotizing mediastinitis (Horowitz, Sosa, & Lickstein) 1990;50:859–60,CP
Sclerosing mediastinitis: improved management with histoplasmosis titer and ketoconazole (Urschel, Razzuk, & others) 1990;50:215–21*

Mediastinum
Acute symptoms due to a huge duplication cyst ruptured into the esophagus (Nahakara, Fujii, & others) 1990;50:309–11,CR
Computed tomography in selecting patients for mediastinoscopy (Van Schil) 1990;50:163,CP; Reply (Thermann) 1990;50:163,CP
Diagnostic imaging and surgical treatment of dumbbell tumors of the mediastinum (Ricci, Rendina, & others) 1990;50:586–9*
Efficacy and benefit of mediastinal computed tomography (Gaer & Goldstraw) 1990;50:508,CP
Esophagobronchial fistula and mediastinal tuberculosis (Raghu & Dillard) 1990;50:647–9,CR
Extended en bloc resection of a primary mediastinal parathyroid carcinoma (Putnam, Schantz, & others) 1990;50:138–40,CR
Left superior vena cava: a pitfall in computed tomographic diagnosis with surgical implications (Wong & Goldstraw) 1990;50:656–7,CR
Mediastinal hibernoma, a rare tumor (Ahn & Harvey) 1990;50:828–30,CR
Primary mediastinal leiomyoma (Shaffer, Pugatch, & Sugarbaker) 1990;50:301–2,CR
Retrotracheal mediastinal goiter with contralateral extension (Ribet) 1990;50:684,CP; Reply (Van Schil) 1990;50:684,CP
Silastic prothesis plombage for right postpneumonectomy syndrome (Riveron, Adams, & others) 1990;50:465–6,CR

Medtronic-Hall valve
Clinical experience with the Medtronic-Hall valve prosthesis (Keenan, Armitage, & others) 1990;50:748–53*
Clinical performance of St. Jude and Medtronic-Hall prostheses: a randomized comparative study (Antunes) 1990;50:743–7*
Eight years' experience with the Medtronic-Hall valve prosthesis (Vallejo, Gonzalez-Santos, & others) 1990;50:429–36*
The Medtronic-Hall valve: frequently used, seldom reported (Starek) 1990;50:346–7,ED

Membrane
Membranotomy for Budd-Chiari syndrome (Victor, Jayanthi, & Madanagopalan) 1990;50:512–3,CP

Mesothelioma
Intraoperative photodynamic therapy for malignant mesothelioma (Pass, Tochner, & others) 1990;50:687–8,CP

Metastases
Squamous carcinoma metastatic to the sternum (D'Orazio, Naunheim, & others) 1990;50:984–6,CR
Strategy for lymphadenectomy in lung cancer three centimeters or less in diameter (Ishida, Yano, & others) 1990;50:708–13*
Surgical management of pulmonary metastatic leiomyosarcoma with gross endobronchial extension (Warren, Bleck, & others) 1990;50:739–42*

Mitral valve
Artificial mitral valve chordae: experimental and clinical experience (Zussa, Frater, & others) 1990;50:367–73*
Biatrial transseptal approach in case of difficult exposure to the mitral valve (Deloche, Acar, & others) 1990;50:318–9,HW
Circumflex artery ventricular fistula and pseudoaneurysm after mitral reoperation (Sutter, Goldman, & others) 1990;50:826–7,CR
Influence of prosthetic design on durability of the Liotta porcine valve in the mitral position (Bortolotti, Milano, & others) 1990;50:734–8*
Mitral prosthetic replacement in small left atria (Campanella, Cameron, & Feilberg) 1990;50:836–7,HW
Polytetrafluoroethylene posterior annuloplasty for mitral regurgitation (Fundarò, Salati, & others) 1990;50:165–6,CP; Reply (Cosgrove) 1990;50:166,CP
Repair of flail anterior leaflets of tricuspid and mitral valves by cusp remodeling (Sutlic, Schmid, & Borst) 1990;50:927–30*
Severe hemolysis after incomplete mitral valve repair (Wilson, Rath, & others) 1990;50:136–7,CR
Ultrasonic debridement during mitral valve reconstruction for calcified mitral stenosis (Nakano, Mitsuno, & others) 1990;50:923–6*

Monitoring
Operative insertion of a transvenous left atrial pressure monitoring line (Satur, Stubington, & others) 1990;50:1004–5,HW
Selective monitoring of patients with suspected blunt cardiac injury (Reif, Justice, & others) 1990;50:530–3*

Morbidity
Pleuropulmonary morbidity: internal thoracic artery versus saphenous vein graft (Hurlbut, Myers, & others) 1990;50:959–64*

Mortality
Clinical experience with the Medtronic-Hall valve prosthesis (Keenan, Armitage, & others) 1990;50:748–53*
Clinical performance of St. Jude and Medtronic-Hall prostheses: a randomized comparative study (Antunes) 1990;50:743–7*
Cor triatriatum: clinical presentation and surgical results in 12 patients (Rodefeld, Brown, & others) 1990;50:562–8*
Critical aortic stenosis in the first month of life: surgical results in 26 infants (Karl, Sano, & others) 1990;50:105–9*
Extension of donor criteria in cardiac transplantation: surgical risk versus supply-side economics (Sweeney, Lammermeier, & others) 1990;50:7–11*
Late results of aortic valvotomy for congenital valvar aortic stenosis (DeBoer, Robbins, & others) 1990;50:69–73*

Long-term results of operation for non–small cell lung cancer in the elderly (Ishida, Yokoyama, & others) 1990;50:919–22*
Relief of tracheal compression by aortopexy (Clevenger, Othersen, & Smith) 1990;50:524–9*
Reoperations on heart valve prostheses: an analysis of operative risks and late results (Pansini, Ottino, & others) 1990;50:590–6*
Results in 104 patients undergoing bronchoplastic procedures for bronchial lesions (Watanabe, Shimizu, & others) 1990;50:607–14*
Severe aortic stenosis in octogenarians: is operation an acceptable alternative? (Deleuze, Loisance, & others) 1990;50:226–9*
Uncomfortable issues (Edmunds) 1990;50:173–4,ED

Multivariate analysis
Analysis of prognostic factors and clinicopathological staging of thymoma (Pescarmona, Rendina, & others) 1990;50:534–8*

Myasthenia gravis
Analysis of prognostic factors and clinicopathological staging of thymoma (Pescarmona, Rendina, & others) 1990;50:534–8*
Coexisting thymic carcinoid tumor and thymoma (Mizuno, Masaoka, & others) 1990;50:650–2,CR
Recurrent thymoma in patients with myasthenia gravis (Ohmi & Ohuchi) 1990;50:243–7*
Thymectomy for myasthenia gravis (Jaretski) 1990;50:686,CP; Reply (Mulder) 1990;50:687,CP

Mycoses
Management of mycotic rupture of the ascending aorta after heart-lung transplantation (Albes, Haverich, & others) 1990;50:982–3,CR

Myocardial infarction
Postinfarction ventricular septal defect repair: retrospective thoughts and historical perspectives (Daggett) 1990;50:1006–9,CL

Myocardial revascularization
Combined internal mammary artery graft for coronary artery revascularization (Bakay, Akçevin, & others) 1990;50:553–6*
Five-year results of coronary bypass grafting for patients older than 70 years: role of internal mammary artery (Azariades, Fessler, & others) 1990;50:940–5*
Internal mammary artery-to-pulmonary artery shunt in infants (Sakakibara, Hori & others) 1990;50:857–8,CP
Selective and adjustable pericardial flap to protect internal mammary artery grafts (González-Santos, Bastida, & others) 1990;50:995–7,HW

Myocardium
Cardioplegia-induced damage to ischemic immature myocardium is independent of oxygen availability (Baker, Boerboom, & Olinger) 1990;50:934–9*
Improved myocardial preservation during cold storage using substrate enhancement (Haan, Lazar, & others) 1990;50:80–5*
Myocardial preservation using lidocaine blood cardioplegia (Fiore, Naunheim, & others) 1990;50:771–5*
Myocardial protection by retrograde cardioplegia in arterial switch operation (Yonenaga, Yasui, & others) 1990;50:238–42*
Preservation of myocardial function and biochemistry after blood and oxygenated crystalloid cardioplegia during cardiac arrest (Coetzee, Roussouw, & others) 1990;50:230–7*

Myxoma
Surgical treatment of cardiac myxomas: long-term results (Sellke, Lemmer, & others) 1990;50:557–61*

Neck
Pharyngoesophageal perforation after blunt neck trauma (Niezgoda, McMenamin, & Graeber) 1990;50:615–7*

Neoplasms
Behavior of small bronchial carcinomas (Shields) 1990;50:691–2,ED
Carcinoid tumors of the thymus (Economopoulos, Lewis, & others) 1990;50:58–61*
Cardiopulmonary cystosarcoma (Shuman) 1990;50:510,CP; Reply (Tyers) 1990;50:510–11,CP
Chest wall recurrence of lung cancer after transthoracic fine needle aspiration biopsy (Hix) 1990;50:1020–1,CP
Coexisting thymic carcinoid tumor and thymoma (Mizuno, Masaoka, & others) 1990;50:650–2,CR
Congenital tracheoesophageal fistula associated with carcinoma of the lung in an adult (Campbell & Salisbury) 1990;50:978–9,CR
Diagnostic imaging and surgical treatment of dumbbell tumors of the mediastinum (Ricci, Rendina, & others) 1990;50:586–9*
Extended en bloc resection of a primary mediastinal parathyroid carcinoma (Putnam, Schantz, & others) 1990;50:138–40,CR
Fatal ectopic thymoma (Jansen & Johnson) 1990;50:469–70,CR
Giant chest wall tumor resulting from tissue reaction to foreign bodies (Fukai, Sano, & others) 1990;50:754–6*
Intramural ventricular fibroma in infancy: survival after partial excision in 2 patients (Ceithaml, Midgley, & others) 1990;50:471–2,CR
Intrathoracic tumors of the vagus nerve (Dabir, Piccione, & Kittle) 1990;50:494–7,CT
Long-term results of operation for non–small cell lung cancer in the elderly (Ishida, Yokoyama, & others) 1990;50:919–22*
Malignant fibrous histiocytoma (Laas & Schmid) 1990;50:164, CP; Reply (Lazar) 1990;50:165,CP; (Craddock) 1990;50:165,CP
Malignant fibrous histiocytoma of the trachea (Randleman, Unger, & Mansour) 1990;50:458–9,CR
Malignant thymoma presenting as intracardiac tumor and superior vena caval obstruction (Airan, Sharma, & others) 1990;50:989–91,CR
Mediastinal hibernoma, a rare tumor (Ahn & Harvey) 1990;50:828–30,CR
Neodymium:YAG laser and brachytherapy in the management of inoperable bronchogenic carcinoma (Miller & Phillips) 1990;50:190–6*
Platysma musculocutaneous flap for reconstruction of trachea in esophageal cancer (Sodeyama, Matsuo, & others) 1990;50:485–7,HW
Recurrent thymoma in patients with myasthenia gravis (Ohmi & Ohuchi) 1990;50:243–7*
Resection of the superior vena cava for primary lung cancer: 5 years' survival (Inoue, Shohtsu, & others) 1990;50:661–2,CR
Results in 104 patients undergoing bronchoplastic procedures for bronchial lesions (Watanabe, Shimizu, & others) 1990;50:607–14*
Role of postoperative radiation therapy in stage IIIa non–small cell lung cancer (Astudillo & Conill) 1990;50:618–23*
Squamous carcinoma metastatic to the sternum (D'Orazio, Naunheim, & others) 1990;50:984–6,CR

Neoplasms (cont'd)
- Strategy for lymphadenectomy in lung cancer three centimeters or less in diameter (Ishida, Yano, & others) 1990;50:708–13*
- Successful Fontan-type operation for a nonresectable right ventricular tumor (Dietl) 1990;50:814–6,CR
- Superior vena caval reconstruction using autologous pericardium (Piccione, Faber, & Warren) 1990;50:417–9*
- Symposium on thoracic surgical oncology 1990;50:500–7,RE
- Use of the centrifugal flow pump for vena caval shunting (Moggio, Praeger, & others) 1990;50:146–8,HW
- Vagal schwannoma (Heitmiller, Labs, & Lipsett) 1990;50:811–3,CR

Neoplastic endocrine-like syndromes
- Bronchial and thymic carcinoids and the ectopic ACTH syndrome (Odell) 1990;50:5–6,ED
- Management of the ectopic ACTH syndrome due to thoracic carcinoids (Pass, Doppman, & others) 1990;50:52–7*

Nerve
- Left recurrent laryngeal nerve paralysis: an unusual presentation of histoplasmosis (Gilbert, Murray, & others) 1990;50:987–8,CR

Neurons
- Modification of supraventricular tachyarrhythmias by stimulating atrial neurons (Ali, Butler, & others) 1990;50:251–6*

Neutrophils
- Complement activation and lung permeability during cardiopulmonary bypass (Tennenberg, Clardy, & others) 1990;50:597–601*

Omentum
- Aortobronchial fistula after an aortic operation (Ishizaki, Tada, & others) 1990;50:975–7,CR
- Management of aortobronchial fistula with graft replacement and omentopexy (Paull & Keagy) 1990;50:972–4,CR
- Use of pedicled omental flap in treatment of empyema (Shirakusa, Ueda, & others) 1990;50:420–4*

Oncology
- Symposium on thoracic surgical oncology 1990;50:500–7,RE

Organ preservation
- Endothelial cell toxicity of solid-organ preservation solutions (von Oppell, Pfeiffer, & others) 1990;50:902–10*

Organ procurement
- Organ procurement for pulmonary transplantation (Ribet, Pruvot) 1990;50:335,CP; Reply (Griffith) 1990;50:335–6,CP

Oxygen
- Cardioplegia-induced damage to ischemic immature myocardium is independent of oxygen availability (Baker, Boerboom, & Olinger) 1990;50:934–9*
- Oxygen free radical scavengers decrease reperfusion injury in lung transplantation (Detterbeck, Keagy, & others) 1990;50:204–10*
- Oxygenated cardioplegia: blood is a many splendored thing (Buckberg) 1990;50:175–7,ED
- Preservation of myocardial function and biochemistry after blood and oxygenated crystalloid cardioplegia during cardiac arrest (Coetzee, Roussouw, & others) 1990;50:230–7*
- Prevention of paraplegia during aortic operations (Wadouh, Wadouh, & others) 1990;50:543–52*

Oxygenation
- Improved cannulation method for extracorporeal membrane oxygenation (Read, St. Cyr, & others) 1990;50:670–1,HW
- Prognostic determinants in extracorporeal membrane oxygenation for respiratory failure in newborns (Weber, Connors, & others) 1990;50:720–3*

Oxygenator
- Blood conservation in cardiac surgery (Scott, Kessler, & Wernly) 1990;50:843–51,CV
- Complement activation with bubble and membrane oxygenators in aortocoronary bypass grafting (Videm, Fosse, & others) 1990;50:387–91*
- Oxygenator failure (Crowley & Brown) 1990;50:859–60,CP

Pain
- Continuous epidural hydromorphone for postthoracotomy pain relief (Brodsky, Chaplan, & others) 1990;50:888–93*

Paralysis
- Diaphragmatic plication (Ribet) 1990;50:855–6,CP
- Left recurrent laryngeal nerve paralysis: an unusual presentation of histoplasmosis (Gilbert, Murray, & others) 1990;50:987–8,CR

Paraplegia
- Paraplegia associated with thoracotomy (Walker) 1990;50:178,ED
- Paraplegia associated with the use of oxidized cellulose in posterolateral thoracotomy incisions (Short) 1990;50:288–90*
- Prevention of paraplegia during aortic operations (Wadouh, Wadouh, & others) 1990;50:543–52*

Parathyroid
- Acute hypercalcemic crisis after an open heart operation (Siclari, Herrmann, & Dralle) 1990;50:831–2,CR
- Extended en bloc resection of a primary mediastinal parathyroid carcinoma (Putnam, Schantz, & others) 1990;50:138–40,CR

Perfusion
- Certification of perfusionists (Fosburg, Anderson, & others) 1990;50:508,CP
- Easy cardioplegia delivery, aortic venting, and reperfusion technique (Kauten) 1990;50:833–5,HW
- Role of perfusion pressure and flow in major organ dysfunction after cardiopulmonary bypass (Slogoff, Reul, & others) 1990;50:911–8*

Pericardium
- Easy dissection of hard and thickened pericardium on constrictive pericarditis (Oku, Sawada, & others) 1990;50:998–9,HW
- Modified pericardial closure to protect cardiovascular structures during sternal reentery (Zapolanski, Fishman, & others) 1990;50:665–6,HW
- Pericardial window: mechanisms of efficacy (Sugimoto, Little, & others) 1990;50:442–5*
- Selective and adjustable pericardial flap to protect internal mammary artery grafts (González-Santos, Bastida, & others) 1990;50:995–7,HW
- Superior vena caval reconstruction using autologous pericardium (Piccione, Faber, & Warren) 1990;50:417–9*

Permeability
- Complement activation and lung permeability during cardiopulmonary bypass (Tennenberg, Clardy, & others) 1990;50:597–601*
- Vein graft permeability (Kennedy, Tedgui, & Piwnica) 1990;50:334,CP; Reply (Finck, Mashburn, & others) 1990;50:334,CP

Pharmacology
- Pharmacological response of internal mammary artery and gastroepiploic artery (Koike, Suma, & others) 1990;50:384–6*

Pharynx
Pharyngoesophageal perforation after blunt neck trauma (Niezgoda, McMenamin, & Graeber) 1990;50:615–7*

Photochemotherapy
Intraoperative photodynamic therapy for malignant mesothelioma (Pass, Tochner, & others) 1990;50:687–8,CP

Plasmapheresis
Acute preoperative plasmapheresis and established blood conservation techniques (Boldt, Kling, & others) 1990;50: 62–8*

Plastics
Air contamination in open heart surgery with disposable coveralls, gowns, and drapes (Verkkala, Mäkelä, & others) 1990;50:757–61*
Plastic struts for delayed sternal closure (Majid) 1990;50: 1021,CP

Platysma
Platysma musculocutaneous flap for reconstruction of trachea in esophageal cancer (Sodeyama, Matsuo, & others) 1990; 50: 485–7,HW

Pleura
Pleuropulmonary morbidity: internal thoracic artery versus saphenous vein graft (Hurlbut, Myers, & others) 1990;50: 959–64*
Thoracoplasty: current application to the infected pleural space (Horrigan & Snow) 1990;50:695–9*

Pneumonectomy
Individual ligation technique (Meyer) 1990;50:1023,CP
Right pneumonectomy syndrome in infancy treated with an expandable prosthesis (Rasch, Grover, & others) 1990;50: 127–9,CR
Silastic prothesis plombage for right postpneumonectomy syndrome (Riveron, Adams, & others) 1990;50:465–6,CR

Pneumopericardium
Tension pneumopericardium as a complication of single-lung transplantation (Cochrane, Mitchell, & others) 1990;50: 808–10,CR

Pneumothorax
Thoracoscopy and talc poudrage for pneumothoraces and effusions (Daniel, Tribble, & Rodgers) 1990;50:186–9*
Thoracoscopic treatment of spontaneous pneumothorax using carbon dioxide laser (Wakabayashi, Brenner, & others) 1990;50:786–90*

Poliomyelitis
A practical mechanical respirator, 1929: the "iron lung" (Meyer) 1990;50:490–3,CL

Polydioxanone
Results of aortic anastomoses made under tension using polydioxanone suture (Haluck, Richenbacher, & others) 1990; 50:392–5*

Polytetrafluoroethylene
Artificial mitral valve chordae: experimental and clinical experience (Zussa, Frater, & others) 1990;50:367–73*
Experimental study of a new porous tracheal prosthesis (Jorge, Ramos, & others) 1990;50:281–7*
Polytetrafluoroethylene graft for spontaneous coronary dissection: 7-year follow-up (McCullough, Abel, & Rubenstein) 1990;50:663–4,CR
Polytetrafluoroethylene posterior annuloplasty for mitral regurgitation (Fundarò, Salati, & others) 1990;50:165–6,CP; Reply (Cosgrove) 1990;50:166,CP
Tracheal reconstruction with polytetrafluoroethylene graft in dogs (Cull, Lally, & others) 1990;50:899–901*

Postoperative care
Continuous epidural hydromorphone for postthoracotomy pain relief (Brodsky, Chaplan, & others) 1990;50:888–93*
Postirradiation pericardectomy (Eckstein) 1990;50:685,CP
Postoperative drainage and pericardial effusion (Bryan & Angelini) 1990;50:330,CP; Reply (Smulders, Moulijn, & Koolen) 1990;50:330,CP
Role of postoperative radiation therapy in stage IIIa non–small cell lung cancer (Astudillo & Conill) 1990;50:618–23*
Timing of operation for aortic regurgitation: relation to postoperative contractile state (Taniguchi, Nakano, & others) 1990;50:779–85*
Treatment of postoperative infection of ascending aorta and transverse aortic arch, including use of viable omentum and muscle flaps (Coselli, Crawford, & others) 1990;50: 868–81*

Postoperative complications
Air contamination in open heart surgery with disposable coveralls, gowns, and drapes (Verkkala, Mäkelä, & others) 1990;50:757–61*
Choreoathetosis after deep hypothermia without circulatory arrest (DeLeon, Ilbawi, & others) 1990;50:714–9*
Complications after cardiac operations in patients with severe pulmonary impairment (Bevelaqua, Garritan, & others) 1990;50:602–6*
Long-term results of operation for non–small cell lung cancer in the elderly (Ishida, Yokoyama, & others) 1990;50:919–22*
Role of perfusion pressure and flow in major organ dysfunction after cardiopulmonary bypass (Slogoff, Reul, & others) 1990;50:911–8*

Postoperative period
Cardiac transplantation (Griffith) 1990;50:161–2,KR
Disruption of a modified Blalock-Taussig shunt by rapid deceleration injury (Joyce & McGrath) 1990;50:124,CR
Long-term results of total repair of tetralogy of Fallot in childhood (Horneffer, Zahka, & others) 1990;50:179–85*
Low-dose preoperative aspirin therapy, postoperative blood loss, and transfusion requirements (Taggart, Siddiqui, & Wheatley) 1990;50:425–8*

Preoperative care
Acute preoperative plasmapheresis and established blood conservation techniques (Boldt, Kling, & others) 1990;50: 62–8*
Initial report of the Veterans Administration preoperative risk assessment study for cardiac surgery (Grover, Hammermeister, & others) 1990;50:12–28*
Low-dose preoperative aspirin therapy, postoperative blood loss, and transfusion requirements (Taggart, Siddiqui, & Wheatley) 1990;50:425–8*
Management of Jehovah's Witness patients (Kyger & Blakestad) 1990;50:167,CP

Pressure
Operative insertion of a transvenous left atrial pressure monitoring line (Satur, Stubington, & others) 1990;50:1004–5,HW

Prognosis
Analysis of prognostic factors and clinicopathological staging of thymoma (Pescarmona, Rendina, & others) 1990;50: 534–8*
Prognostic determinants in extracorporeal membrane oxygenation for respiratory failure in newborns (Weber, Connors, & others) 1990;50:720–3*
Pulmonary resection combined with cardiac operations (Canver, Bhayana, & others) 1990;50:796–9*

Prognosis (cont'd)
  Surgical treatment of cardiac myxomas: long-term results (Sellke, Lemmer, & others) 1990;50:557–61*
Prophylaxis
  Antimicrobial prophylaxis for open heart operations (Miedzinski, Callaghan, & others) 1990;50:800–7*
  Prophylactic digitalization for thoracotomy: a reassessment (Ritchie, Bowe, & Gibbons) 1990;50:86–8*
Prostaglandins
  Reversal of protamine-induced catastrophic pulmonary vasoconstriction by prostaglandin E_1 (Whitman, Martel, & others) 1990;50:303–5,CR
Prosthesis
  Catastrophic thrombosis of porcine aortic bioprostheses (Baciewicz, del Rio, & others) 1990;50:817–9,CR
  Clinical experience with the Medtronic-Hall valve prosthesis (Keenan, Armitage, & others) 1990;50:748–53*
  Clinical performance of St. Jude and Medtronic-Hall prostheses: a randomized comparative study (Antunes) 1990;50:743–7*
  Eight years' experience with the Medtronic-Hall valve prosthesis (Vallejo, Gonzalez-Santos, & others) 1990;50:429–36*
  Experimental study of a new porous tracheal prosthesis (Jorge, Ramos, & others) 1990;50:281–7*
  Influence of prosthetic design on durability of the Liotta porcine valve in the mitral position (Bortolotti, Milano, & others) 1990;50:734–8*
  Long-term prosthesis-related and sudden cardiac-related complications after valve replacement for aortic stenosis (Lund, Pilegaard, & others) 1990;50:396–406*
  Mitral prosthetic replacement in small left atria (Campanella, Cameron, & Feilberg) 1990;50:836–7,HW
  Reoperations on heart valve prostheses: an analysis of operative risks and late results (Pansini, Ottino, & others) 1990;50:590–6*
  Right pneumonectomy syndrome in infancy treated with an expandable prosthesis (Rasch, Grover, & others) 1990;50:127–9,CR
  Silastic prothesis plombage for right postpneumonectomy syndrome (Riveron, Adams, & others) 1990;50:465–6,CR
  Use of the variable-length intraluminal sutureless graft (Graver & Tyras) 1990;50:624–30*
Pulmonary
  Acquired benign esophagorespiratory fistula: report of 16 consecutive cases (Gerzić, Rakić, & Randjelović) 1990;50:724–7*
  Bilateral pulmonary nodules caused by *Dirofilaria immitis* (Bradham, Locklair, & Grimball) 1990;50:312–3,CR
  Cardiopulmonary cystosarcoma (Shuman) 1990;50:510,CP; Reply (Tyers) 1990;50:510–11,CP
  Combined repair of transposed great arteries and total anomalous pulmonary venous connection (Amodeo, Corno, & others) 1990;50:820–1,CR
  Complement activation and lung permeability during cardiopulmonary bypass (Tennenberg, Clardy, & others) 1990;50:597–601*
  Complications after cardiac operations in patients with severe pulmonary impairment (Bevelaqua, Garritan, & others) 1990;50:602–6*
  Decreased vascular compliance after reimplantation of the left lower lobe in young pigs (Johnson, Teague, & others) 1990;50:277–80*
  Determinants of pulmonary function in patients undergoing coronary bypass operations (Shapira, Zabatino, & others) 1990;50:268–73*
  Feasibility of reversible pulmonary artery banding: early results and intermediate-term follow-up (Epstein, Duncan, & others) 1990;50:94–7*
  Individual ligation technique (Meyer) 1990;50:1023,CP
  Internal mammary artery graft to pulmonary vasculature fistula: a cause of recurrent angina (Johnson, Schmaltz, & others) 1990;50:297–8,CR
  Intraoperative bronchial aspiration of ruptured pulmonary hydatid cysts (Saidi & Rezvan-Nobahar) 1990;50:631–6*
  Long-term results of operation for non–small cell lung cancer in the elderly (Ishida, Yokoyama, & others) 1990;50:919–22*
  Long-term results of total repair of tetralogy of Fallot in childhood (Horneffer, Zahka, & others) 1990;50:179–85*
  Morphology of pulmonary atresia with intact ventricular septum in patients dying after operation (van de Wal, Smith, & others) 1990;50:90–102*
  New approach to the surgical treatment of Swyer-James-MacLeod syndrome (Vishnevsky & Nikoladze) 1990;50:103–4*
  Pleuropulmonary morbidity: internal thoracic artery versus saphenous vein graft (Hurlbut, Myers, & others) 1990;50:959–64*
  Pulmonary resection combined with cardiac operations (Canver, Bhayana, & others) 1990;50:796–9*
  Reversal of protamine-induced catastrophic pulmonary vasoconstriction by prostaglandin E_1 (Whitman, Martel, & others) 1990;50:303–5,CR
  Reversibility of pulmonary artery hypertension in aortic stenosis after aortic valve replacement (Tracy, Proctor, & Hizny) 1990;50:89–93*
  Scimitar syndrome: cine magnetic resonance imaging demonstration of anomalous pulmonary venous drainage (Baxter, McFadden, & others) 1990;50:121–3,CR
  Strategy for lymphadenectomy in lung cancer three centimeters or less in diameter (Ishida, Yano, & others) 1990;50:708–13*
  Surgical management of pulmonary metastatic leiomyosarcoma with gross endobronchial extension (Warren, Bleck, & others) 1990;50:739–42*
  Thoracoplasty: current application to the infected pleural space (Horrigan & Snow) 1990;50:695–9*
  Thoracoscopy and talc poudrage for pneumothoraces and effusions (Daniel, Tribble, & Rodgers) 1990;50:186–9*
  Thoracoscopic treatment of spontaneous pneumothorax using carbon dioxide laser (Wakabayashi, Brenner, & others) 1990;50:786–90*
  A useful technique for closure of pulmonary lacerations (Fulton & Dickson) 1990;50:149–50,HW
Pulmonary artery
  Efficacy of pulmonary artery versus left ventricular venting (Garibaldi) 1990;50:685,CP; Reply (Mitchell) 1990;50:686,CP
  Swan-Ganz catheter-induced massive hemoptysis and pulmonary artery false aneurysm (Feng, Singh, & others) 1990;50:644–6,CR
Pumps
  Beware centrifugal pumps: not a one-way street, but a potentially dangerous "siphon" (Kolff, McClurken, & Alpern) 1990;50:512,CP
  Origins of intraaortic balloon pumping (Kantrowitz) 1990;50:672–4,CL
  Rapid placement of the Hemopump and hemofiltration cannula (Duncan, Burnett, & others) 1990;50:667–9,HW
  Use of the centrifugal flow pump for vena caval shunting (Moggio, Praeger, & others) 1990;50:146–8,HW

Punctures
Risks of blood exposure to the cardiac surgical team (Pate) 1990;50:248–50*

Rabbit
Cardioplegia-induced damage to ischemic immature myocardium is independent of oxygen availability (Baker, Boerboom, & Olinger) 1990;50:934–9*
Experimental study of a new porous tracheal prosthesis (Jorge, Ramos, & others) 1990;50:281–7*
Improved myocardial preservation during cold storage using substrate enhancement (Haan, Lazar, & others) 1990;50: 80–5*

Radiation
Postirradiation pericardectomy (Eckstein) 1990;50:685,CP
Role of postoperative radiation therapy in stage IIIa non-small cell lung cancer (Astudillo & Conill) 1990;50:618–23*

Rats
Long-term amiodarone administration protects against global myocardial ischemia (Karlson, Hopkins, & others) 1990;50: 575–8*

Regurgitation
Timing of operation for aortic regurgitation: relation to postoperative contractile state (Taniguchi, Nakano, & others) 1990;50:779–85*

Reimplantation
Decreased vascular compliance after reimplantation of the left lower lobe in young pigs (Johnson, Teague, & others) 1990;50:277–80*

Renal
Internal mammary artery revascularization in the patient on long-term renal dialysis (Blakeman, Sullivan, & others) 1990;50:776–8*

Reoperation
Circumflex artery ventricular fistula and pseudoaneurysm after mitral reoperation (Sutter, Goldman, & others) 1990;50: 826–7,CR
Critical aortic stenosis in the first month of life: surgical results in 26 infants (Karl, Sano, & others) 1990;50:105–9*
Management of mycotic rupture of the ascending aorta after heart-lung transplantation (Albes, Haverich, & others) 1990;50:982–3,CR
Reappraisal of localized resection for subvalvar aortic stenosis (Stewart, Merrill, & others) 1990;50:197–203*
Reoperations on heart valve prostheses: an analysis of operative risks and late results (Pansini, Ottino, & others) 1990;50:590–6*

Report
Symposium on thoracic surgical oncology 1990;50:500–7,RE

Respiration
Prognostic determinants in extracorporeal membrane oxygenation for respiratory failure in newborns (Weber, Connors, & others) 1990;50:720–3*
Respiratory function after thoracic operations (Hallfeldt, Knoefel, & others) 1990;50:688,CP

Respirators
A practical mechanical respirator, 1929: the "iron lung" (Meyer) 1990;50:490–3,HI

Resuscitation
Extension of donor criteria in cardiac transplantation: surgical risk versus supply-side economics (Sweeney, Lammermeier, & others) 1990;50:7–11*

Revascularization
Alternate method for revascularization of the posterior lateral coronary artery (Bolling & Kirsh) 1990;50:838,HW
Coronary revascularization in a child with Kawasaki disease: use of right gastroepiploic artery (Takeuchi, Gomi, & others) 1990;50:294–6,CR
Easy cardioplegia delivery, aortic venting, and reperfusion technique (Kauten) 1990;50:833–5,HW
Internal mammary artery revascularization in the patient on long-term renal dialysis (Blakeman, Sullivan, & others) 1990;50:776–8*
Modified pericardial closure to protect cardiovascular structures during sternal reentery (Zapolanski, Fishman, & others) 1990;50:665–6,HW
Myocardial preservation using lidocaine blood cardioplegia (Fiore, Naunheim, & others) 1990;50:771–5*
Surgical standby for coronary angioplasty (Ullyot) 1990;50:3–4,ED

Risk factors
Five-year results of coronary bypass grafting for patients older than 70 years: role of internal mammary artery (Azariades, Fessler, & others) 1990;50:940–5*
Initial report of the Veterans Administration preoperative risk assessment study for cardiac surgery (Grover, Hammermeister, & others) 1990;50:12–28*
Risks of blood exposure to the cardiac surgical team (Pate) 1990;50:248–50*
Severe aortic stenosis in octogenarians: is operation an acceptable alternative? (Deleuze, Loisance, & others) 1990;50: 226–9*
Uncomfortable issues (Edmunds) 1990;50:173–4,ED

Sarcoma
Surgical management of pulmonary metastatic leiomyosarcoma with gross endobronchial extension (Warren, Bleck, & others) 1990;50:739–42*

Scavengers
Oxygen free radical scavengers decrease reperfusion injury in lung transplantation (Detterbeck, Keagy, & others) 1990; 50:204–10*

Schwannoma
Vagal schwannoma (Heitmiller, Labs, & Lipsett) 1990;50:811–3,CR

Scimitar syndrome
Scimitar syndrome: cine magnetic resonance imaging demonstration of anomalous pulmonary venous drainage (Baxter, McFadden, & others) 1990;50:121–3,CR

Sclerosing
Sclerosing mediastinitis: improved management with histoplasmosis titer and ketoconazole (Urschel, Razzuk, & others) 1990;50:215–21*

Sheep
Artificial mitral valve chordae: experimental and clinical experience (Zussa, Frater, & others) 1990;50:367–73*

Shock
Effects of left ventricular assist for cardiogenic shock on cardiac function and organ blood flow distribution (Sukehiro & Flameng) 1990;50:374–83*
Improved cannulation method for extracorporeal membrane oxygenation (Read, St. Cyr, & others) 1990;50:670–1,HW

Shunt
Disruption of a modified Blalock-Taussig shunt by rapid deceleration injury (Joyce & McGrath) 1990;50:124,CR
Liver transplantation with atrioatrial anastomosis for Budd-Chiari syndrome (Carrel, Decurtins, & others) 1990;50: 658–60,CR
Superior vena caval reconstruction using autologous pericardium (Piccione, Faber, & Warren) 1990;50:417–9*

Silicone elastomers
Silastic prothesis plombage for right postpneumonectomy syndrome (Riveron, Adams, & others) 1990;50:465–6,CR
Smoking
Long-term results of operation for non–small cell lung cancer in the elderly (Ishida, Yokoyama, & others) 1990;50:919–22*
The Society of Thoracic Surgeons
Program, 1990 Interim Meeting, 1990;50:168–72;338–42
Twenty-seventh Annual Meeting program 1990;50:1024–35
The Southern Thoracic Surgical Association
Program, Thirty-seventh Annual Meeting 1990;50:515–8
Spinal cord
Prevention of paraplegia during aortic operations (Wadouh, Wadouh, & others) 1990;50:543–52*
Spine
Thoracic spinal fractures and aortic rupture: a significant and fatal association (Sturm, Hines, & Perry) 1990;50:931–3*
Spleen
Coronary artery bypass grafting without splenectomy (Bowman) 1990;50:330–1,CP; Reply (Koike) 1990;50:331,CP
Staging
Analysis of prognostic factors and clinicopathological staging of thymoma (Pescarmona, Rendina, & others) 1990;50: 534–8*
Staphylococcus
Antimicrobial prophylaxis for open heart operations (Miedzinski, Callaghan, & others) 1990;50:800–7*
Job's syndrome: a rare cause of recurrent lung abscess in childhood (Lui & Inculet) 1990;50:992–4,CR
Steal
Steal phenomenon caused by a parallel branch of the internal mammary artery (Schmid, Heublein, & others) 1990;50: 463–4,CR
Stenosis
Correction of truncus arteriosus with truncal valvar stenosis or insufficiency using two homografts (Elkins, Steinberg, & others) 1990;50:728–33*
Critical aortic stenosis in the first month of life: surgical results in 26 infants (Karl, Sano, & others) 1990;50:105–9*
Late results of aortic valvotomy for congenital valvar aortic stenosis (DeBoer, Robbins, & others) 1990;50:69–73*
Long-term prosthesis-related and sudden cardiac-related complications after valve replacement for aortic stenosis (Lund, Pilegaard, & others) 1990;50:396–406*
Norwood operation for univentricular heart with subaortic stenosis in the neonate (Tchervenkov, Béland, & others) 1990;50:822–5,CR
Reappraisal of localized resection for subvalvar aortic stenosis (Stewart, Merrill, & others) 1990;50:197–203*
Results of aortic anastomoses made under tension using polydioxanone suture (Haluck, Richenbacher, & others) 1990; 50:392–5*
Reversibility of pulmonary artery hypertension in aortic stenosis after aortic valve replacement (Tracy, Proctor, & Hizny) 1990;50:89–93*
Subvalvar aortic stenosis: timing of operation (Douville & Sade), 1990;50:29–34*
Tubular supravalvar aortic stenosis: replacement of ascending aorta in a young patient (Gargiulo, Zannini, & others) 1990;50:467–8,CR
Ultrasonic debridement during mitral valve reconstruction for calcified mitral stenosis (Nakano, Mitsuno, & others) 1990; 50:923–6*
Uncomfortable issues (Edmunds) 1990;50:173–4,ED
Sternotomy
Sternotomy infection: poor prediction by acute phase response and delayed hypersensitivity (Ulicny, Hiratzka, & others) 1990;50:949–58*
Sternum
Plastic struts for delayed sternal closure (Majid) 1990;50: 1021,CP
Squamous carcinoma metastatic to the sternum (D'Orazio, Naunheim, & others) 1990;50:984–6,CR
Storage
Leukocyte depletion results in excellent heart-lung function after 12 hours of storage (Pillai, Bando, & others) 1990;50: 211–4*
Subaortic
Subaortic obstruction: intraoperative echocardiography as an adjunct to operation (Sreeram, Sutherland, & others) 1990; 50:579–85*
Substrates
Improved myocardial preservation during cold storage using substrate enhancement (Haan, Lazar, & others) 1990;50: 80–5*
Suction
Double-barrel suction device (Robicsek) 1990;50:509,CP
Surgical equipment
Choice of surgical clips for mammary artery take-down (Hovaguimian & Floten) 1990;50:856,CP; Reply (Gold) 1990;50: 856–7,CP
Double-barrel suction device (Robicsek) 1990;50:509,CP
A fiber-optic retractor for harvesting the internal mammary artery (Angelini & Azzu) 1990;50:314–5,HW
Surgical flaps
Platysma musculocutaneous flap for reconstruction of trachea in esophageal cancer (Sodeyama, Matsuo, & others) 1990; 50:485–7,HW
Use of pedicled omental flap in treatment of empyema (Shirakusa, Ueda, & others) 1990;50:420–4*
Survival
Clinical experience with portable cardiopulmonary bypass in cardiac arrest patients (Hartz, LoCicero, & others) 1990;50: 437–41*
Clinical performance of St. Jude and Medtronic-Hall prostheses: a randomized comparative study (Antunes) 1990;50: 743–7*
Eight years' experience with the Medtronic-Hall valve prosthesis (Vallejo, Gonzalez-Santos, & others) 1990;50:429–36*
Intramural ventricular fibroma in infancy: survival after partial excision in 2 patients (Ceithaml, Midgley, & others) 1990; 50:471–2,CR
Long-term results of operation for non–small cell lung cancer in the elderly (Ishida, Yokoyama, & others) 1990;50:919–22*
Morphology of pulmonary atresia with intact ventricular septum in patients dying after operation (van de Wal, Smith, & others) 1990;50:90–102*
Pulmonary resection combined with cardiac operations (Canver, Bhayana, & others) 1990;50:796–9*
Resection of the superior vena cava for primary lung cancer: 5 years' survival (Inoue, Shohtsu, & others) 1990;50:661–2,CR
Results in 104 patients undergoing bronchoplastic procedures for bronchial lesions (Watanabe, Shimizu, & others) 1990; 50:607–14*
Role of postoperative radiation therapy in stage IIIa non–small cell lung cancer (Astudillo & Conill) 1990;50:618–23*

- Strategy for lymphadenectomy in lung cancer three centimeters or less in diameter (Ishida, Yano, & others) 1990;50:708–13*

Sutures
- Results of aortic anastomoses made under tension using polydioxanone suture (Haluck, Richenbacher, & others) 1990;50:392–5*
- Sutureless ring graft replacement of ascending aorta and aortic arch (Oz, Ashton, & others) 1990;50:74–9*

Swine
- Decreased vascular compliance after reimplantation of the left lower lobe in young pigs (Johnson, Teague, & others) 1990;50:277–80*

Swyer-James-MacLeod syndrome
- New approach to the surgical treatment of Swyer-James-MacLeod syndrome (Vishnevsky & Nikoladze) 1990;50:103–4*

Tachycardia
- Modification of supraventricular tachyarrhythmias by stimulating atrial neurons (Ali, Butler, & others) 1990;50:251–6*
- Successful surgical treatment of incessant automatic atrial tachycardia with atrial aneurysm (Miyamura, Nakagomi, & others) 1990;50:476–8,CR

Talc
- Thoracoscopy and talc poudrage for pneumothoraces and effusions (Daniel, Tribble, & Rodgers) 1990;50:186–9*

Temperature
- Choreoathetosis after deep hypothermia without circulatory arrest (DeLeon, Ilbawi, & others) 1990;50:714–9*
- Is systemic cooling essential for open heart surgery? (Victor & Kabeer) 1990;50:334–5,CP

Tetralogy of Fallot
- Long-term results of total repair of tetralogy of Fallot in childhood (Horneffer, Zahka, & others) 1990;50:179–85*
- Repair of complete atrioventricular septal defect with tetralogy of Fallot (Ilbawi, Cua, & others) 1990;50:407–12*
- Variations within the fibrous skeleton and ventricular outflow tracts in tetralogy of Fallot (Howell, Ho, & others) 1990;50:450–7*

Thoracic neoplasms
- Management of the ectopic ACTH syndrome due to thoracic carcinoids (Pass, Doppman, & others) 1990;50:52–7*

Thoracic surgery
- Acquired benign esophagorespiratory fistula: report of 16 consecutive cases (Gerzić, Rakić, & Randjelović) 1990;50:724–7*
- Collis gastroplasty: origin and evolution (Adler) 1990;50:839–42,CL
- Complete foregut duplication (Dresler, Patterson, & others) 1990;50:306–8,CR
- Cor triatriatum: clinical presentation and surgical results in 12 patients (Rodefeld, Brown, & others) 1990;50:562–8*
- Diagnostic imaging and surgical treatment of dumbbell tumors of the mediastinum (Ricci, Rendina, & others) 1990;50:586–9*
- Empyema thoracis (Miller) 1990;50:343–4,ED
- Extended en bloc resection of a primary mediastinal parathyroid carcinoma (Putnam, Schantz, & others) 1990;50:138–40,CR
- Feasibility of reversible pulmonary artery banding: early results and intermediate-term follow-up (Epstein, Duncan, & others) 1990;50:94–7*
- Flow capacity of the human retrograde internal mammary artery: surgical considerations (González-Santos, Bastida, & others) 1990;50:360–6*
- Four years' experience with fibrin sealant in thoracic and cardiovascular surgery (Matthew, Spotnitz, & others) 1990;50:40–4*
- Intrathoracic tumors of the vagus nerve (Dabir, Piccione, & Kittle) 1990;50:494–7,CT
- Management of empyema thoracis (Ali & Unruh) 1990;50:355–9*
- Modified pericardial closure to protect cardiovascular structures during sternal reentery (Zapolanski, Fishman, & others) 1990;50:665–6,HW
- New approach to the surgical treatment of Swyer-James-MacLeod syndrome (Vishnevsky & Nikoladze) 1990;50:103–4*
- Pericardial window: mechanisms of efficacy (Sugimoto, Little, & others) 1990;50:442–5*
- Prevention of paraplegia during aortic operations (Wadouh, Wadouh, & others) 1990;50:543–52*
- Prophylactic digitalization for thoracotomy: a reassessment (Ritchie, Bowe, & Gibbons) 1990;50:86–8*
- Pulmonary resection combined with cardiac operations (Canver, Bhayana, & others) 1990;50:796–9*
- Repair of thoracic dissections (Hammond) 1990;50:330,CP
- Respiratory function after thoracic operations (Hallfeldt, Knoefel, & others) 1990;50:688,CP
- Results in 104 patients undergoing bronchoplastic procedures for bronchial lesions (Watanabe, Shimizu, & others) 1990;50:607–14*
- Retrotracheal goiter: a diagnostic and therapeutic problem (Waldron, Coffey, & others) 1990;50:133–5,CR
- Sternotomy infections in infants (Stahl & Kopf) 1990;50:337,CP; Reply (Terranova & Crawford) 1990;50:337,CP
- Subaortic obstruction: intraoperative echocardiography as an adjunct to operation (Sreeram, Sutherland, & others) 1990;50:579–85*
- Surgical management of chronic empyema: the role of thoracoplasty (Pairolero & Trastek) 1990;50:689–90,ED
- Surgical management of pulmonary metastatic leiomyosarcoma with gross endobronchial extension (Warren, Bleck, & others) 1990;50:739–42*
- Surgical repair of type II truncus arteriosus without a conduit (Sharma, Pilato, & Ott) 1990;50:479–81,HW
- Sutureless ring graft replacement of ascending aorta and aortic arch (Oz, Ashton, & others) 1990;50:74–9*
- Symposium on thoracic surgical oncology 1990;50:500–7,RE
- Tracheal and main bronchial disruptions after blunt chest trauma: presentation and management (Baumgartner, Sheppard, & others) 1990;50:569–74*
- Traumatic bronchial rupture and other major thoracic injuries (Benfield) 1990;50:523,ED
- Use of pedicled omental flap in treatment of empyema (Shirakusa, Ueda, & others) 1990;50:420–4*

Thoracoplasty
- Surgical management of chronic empyema: the role of thoracoplasty (Pairolero & Trastek) 1990;50:689–90,ED
- Thoracoplasty: current application to the infected pleural space (Horrigan & Snow) 1990;50:695–9*

Thoracoscopy
- Thoracoscopy and talc poudrage for pneumothoraces and effusions (Daniel, Tribble, & Rodgers) 1990;50:186–9*
- Thoracoscopic treatment of spontaneous pneumothorax using carbon dioxide laser (Wakabayashi, Brenner, & others) 1990;50:786–90*

Thoracotomy
Acute symptoms due to a huge duplication cyst ruptured into the esophagus (Nahakara, Fujii, & others) 1990;50:309–11,CR
Continuous epidural hydromorphone for postthoracotomy pain relief (Brodsky, Chaplan, & others) 1990;50:888–93*
Paraplegia associated with thoracotomy (Walker) 1990;50:178,ED
Paraplegia associated with the use of oxidized cellulose in posterolateral thoracotomy incisions (Short) 1990;50:288–90*
Thorax
Chest wall recurrence of lung cancer after transthoracic fine needle aspiration biopsy (Hix) 1990;50:1020–1,CP
Giant chest wall tumor resulting from tissue reaction to foreign bodies (Fukai, Sano, & others) 1990;50:754–6*
Thoracic spinal fractures and aortic rupture: a significant and fatal association (Sturm, Hines, & Perry) 1990;50:931–3*
Tracheal and main bronchial disruptions after blunt chest trauma: presentation and management (Baumgartner, Sheppard, & others) 1990;50:569–74*
Thromboembolism
Clinical experience with the Medtronic-Hall valve prosthesis (Keenan, Armitage, & others) 1990;50:748–53*
Thrombosis
Catastrophic thrombosis of porcine aortic bioprostheses (Baciewicz, del Rio, & others) 1990;50:817–9,CR
Thymectomy
Thymectomy for myasthenia gravis (Jaretski) 1990;50:686,CP; Reply (Mulder) 1990;50:687,CP
Thymoma
Analysis of prognostic factors and clinicopathological staging of thymoma (Pescarmona, Rendina, & others) 1990;50:534–8*
Assessing the prognosis of thymomas (Wick) 1990;50:521–2,ED
Coexisting thymic carcinoid tumor and thymoma (Mizuno, Masaoka, & others) 1990;50:650–2,CR
Fatal ectopic thymoma (Jansen & Johnson) 1990;50:469–70,CR
Malignant thymoma presenting as intracardiac tumor and superior vena caval obstruction (Airan, Sharma, & others) 1990;50:989–91,CR
Recurrent thymoma in patients with myasthenia gravis (Ohmi & Ohuchi) 1990;50:243–7*
Thymus
Bronchial and thymic carcinoids and the ectopic ACTH syndrome (Odell) 1990;50:5–6,ED
Carcinoid tumors of the thymus (Economopoulos, Lewis, & others) 1990;50:58–61*
Coexisting thymic carcinoid tumor and thymoma (Mizuno, Masaoka, & others) 1990;50:650–2,CR
Tissue donors
Extension of donor criteria in cardiac transplantation: surgical risk versus supply-side economics (Sweeney, Lammermeier, & others) 1990;50:7–11*
Use of potentially infected donor hearts for cardiac transplantation (Lammermeier, Sweeney, & others) 1990;50:222–5*
Titanium
Choice of surgical clips for mammary artery take-down (Hovaguimian & Floten) 1990;50:856,CP; Reply (Gold) 1990;50:856–7,CP
Tomography
Acute symptoms due to a huge duplication cyst ruptured into the esophagus (Nakahara, Fujii, & others) 1990;50:309–11,CR
Complete foregut duplication (Dresler, Patterson, & others) 1990;50:306–8,CR
Computed tomography in selecting patients for mediastinoscopy (Van Schil) 1990;50:163,CP; Reply (Thermann) 1990;50:163,CP
Efficacy and benefit of mediastinal computed tomography (Gaer & Goldstraw) 1990;50:508,CP
Left superior vena cava: a pitfall in computed tomographic diagnosis with surgical implications (Wong & Goldstraw) 1990;50:656–7,CR
Systemic venous aneurysms (Yokomise, Nakayama, & others) 1990;50:460–2,CR
Toxicity
Endothelial cell toxicity of solid-organ preservation solutions (von Oppell, Pfeiffer, & others) 1990;50:902–10*
Trachea
Congenital tracheoesophageal fistula associated with carcinoma of the lung in an adult (Campbell & Salisbury) 1990;50:978–9,CR
Experimental study of a new porous tracheal prosthesis (Jorge, Ramos, & others) 1990;50:281–7*
Malignant fibrous histiocytoma of the trachea (Randleman, Unger, & Mansour) 1990;50:458–9,CR
Platysma musculocutaneous flap for reconstruction of trachea in esophageal cancer (Sodeyama, Matsuo, & others) 1990;50:485–7,HW
Relief of tracheal compression by aortopexy (Clevenger, Othersen, & Smith) 1990;50:524–9*
Retrotracheal goiter: a diagnostic and therapeutic problem (Waldron, Coffey, & others) 1990;50:133–5,CR
Tracheal and main bronchial disruptions after blunt chest trauma: presentation and management (Baumgartner, Sheppard, & others) 1990;50:569–74*
Tracheal reconstruction with polytetrafluoroethylene graft in dogs (Cull, Lally, & others) 1990;50:899–901*
Transfusion
Aspirin, blood loss, and transfusion (Gay) 1990;50:345,ED
Guidelines for transfusion support in patients undergoing coronary artery bypass grafting (Goodnough, Johnston, & others) 1990;50:675–83,CV
How should the risk of bank blood transfusion in open heart operations be minimized? (Santoli, Scrofani, & Santoli) 1990;50:1021–2,CP
Low-dose preoperative aspirin therapy, postoperative blood loss, and transfusion requirements (Taggart, Siddiqui, & Wheatley) 1990;50:425–8*
Transplantation
Cardiac transplantation (Griffith) 1990;50:161–2,KR
Cardiac transplantation for hypoplastic left heart syndrome: a modified technique (Backer, Idriss, & others) 1990;50:894–8*
Cardiac transplantation for neonates with hypoplastic left heart syndrome (Mayer) 1990;50:864–5,ED
Cardioplegia for transplantation: failure of extracellular solution compared with Stanford or UW solution (Gott, Pan-Chih, & others) 1990;50:348–54*
Extension of donor criteria in cardiac transplantation: surgical risk versus supply-side economics (Sweeney, Lammermeier, & others) 1990;50:7–11*
Improved myocardial preservation during cold storage using substrate enhancement (Haan, Lazar, & others) 1990;50:80–5*
Liver transplantation with atrioatrial anastomosis for Budd-

- Chiari syndrome (Carrel, Decurtins, & others) 1990;50:658–60,CR
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Herbert Sloan, MD

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THE ANNALS OF THORACIC SURGERY

Official Journal of The Society of Thoracic Surgeons and the Southern Thoracic Surgical Association

EVENTS OF INTEREST

Brief notices of meetings, courses, and symposia relevant to cardiothoracic surgery are published as a service. They should be formatted as follows: meeting title, date, place, sponsor, accreditation, director, fee, minimum/maximum attendance, and address/phone for further information. Announcements should be sent at least 4 months in advance of publication to *The Annals of Thoracic Surgery*, 3108 Queeny Tower, Barnes Hospital Plaza, St. Louis, MO 63110-1041; telephone, (314) 361-6084.

Global Plenum on Organ Transplantation, Lucknow, India—December 14–16, 1990

For information on this meeting, contact Prof Probal Ghosh, Plenum Secretariat, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Raebareli Rd, Post Box 375, Lucknow 226001, India.

Esophageal and Foregut Disorders: Pathophysiology and Treatment, Big Island of Hawaii—January 24–29, 1991

For information on this meeting, contact Department of Surgery, Creighton University, CME Program, 601 N 30th St, Omaha, NE 68131; or telephone (402) 280-4501 (facsimile: (402) 280-4593).

Cardiopulmonary Rehabilitation: Status '91, Gainesville, Florida—January 28–30, 1991

For information on this meeting, telephone Michael L. Pollock, PhD, at (904) 392-9575.

Twenty-seventh Annual Meeting of The Society of Thoracic Surgeons, San Francisco, California—February 17–20, 1991

For information on this meeting, contact Richard P. Anderson, MD, The Society of Thoracic Surgeons, 401 N Michigan Ave, Chicago, IL 60611; or telephone (312) 644-6610.

Fortieth Annual Meeting of the American College of Cardiology, Atlanta, Georgia—March 3–7, 1991

For information on this meeting, contact American College of Cardiology, 9111 Old Georgetown Rd, Bethesda, MD 20814; or telephone (800) 253-4636.

First International Symposium on Diseases of the Aorta, Tokyo, Japan—March 6–8, 1991

For information on this meeting, contact Secretariat of ISDA, c/o Japan Convention Services, Inc, Nippon Press Center Bldg, 2-2-1, Uchisaiwai-cho, Chiyoda-ku, Tokyo 100, Japan.

Arrhythmia Surgery Operative Clinics, St. Louis, Missouri—March 21–23, 1991, and October 3–5, 1991

For information on these meetings, contact Beverly Farrier, ProMedica International, 620 Newport Center Dr, Suite 575, Newport Beach, CA 92660; or telephone (714) 640-5870 (facsimile: (714) 759-6911).

Neonatal Cardiac Surgery for Transposition of the Great Arteries, Plessis-Robinson, France—March 24–28, 1991

For information on this meeting, contact Aloma Wybenga, ProMedica International, 620 Newport Center Dr, Suite 575, Newport Beach, CA 92660; or telephone (714) 640-5870 (facsimile: (714) 759-6911).

General Thoracic Surgery, Boston, Massachusetts—April 22–23, 1991

For information on this meeting, contact the Department of Continuing Education, Harvard Medical School, PO Box 825, Boston, MA 02117; or telephone (617) 432-1525.

Myocardial Assist Techniques Seminar I, Washington, DC—May 2–3, 1991

For information on this meeting, contact Margo Irr, ProMedica International, 620 Newport Center Dr, Suite 575, Newport Beach, CA 92660; or telephone (714) 640-5870 (facsimile: (714) 759-6911).

Seventy-first Annual Meeting of The American Association for Thoracic Surgery, Washington, DC—May 6–8, 1991

For information on this meeting, contact American Association for Thoracic Surgery, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Quality of Life After Open Heart Surgery, Antwerp, Belgium—May 16–18, 1991

This meeting is sponsored by The World Health Organization, the European Society of Cardiology, and the International Society and Federation of Cardiology. For information on this meeting, contact P. J. Walter, MD, FACS, Professor and Chairman of the Department of Cardiovascular Surgery, University Clinic of Antwerp, B 2520 Antwerp, Wilrijkstraat 10, Belgium; or telephone 00323.829.11.11 (facsimile: 00323.830.20.99).

Vascular Surgery 1991—Current State of the Art, New York, New York—May 16–18, 1991

For information on this meeting, contact Ann J. Boehme, CMP, Associate Director for Continuing Education, Long Island Jewish Medical Center, New Hyde Park, NY 11042; or telephone (718) 470-8650.

Coronary Surgery: Clinical and Basic Perspectives, Sheffield, England—May 17, 1991

For information on this meeting, contact G. D. Angelini, Clinical Sciences Centre, Northern General Hospital, Herries Rd, Sheffield S5 7AU, England.

Surgery for Coronary Artery Disease, Durham, North Carolina—May 20–21, 1991

For information on this meeting, contact Vanessa Moore, PO Box 2986, Duke University Medical Center, Durham, NC 27710, or telephone (919) 684-6077; or contact American College of Cardiology, Extramural Programs Department, 9111 Old Georgetown Rd, Bethesda, MD 20814, or telephone (800) 253-4636.

Fifth International Symposium on Cardiac Bioprostheses, Avignon, France—May 24–27, 1991

This meeting is sponsored by the Scientific Committee for the Advancement of Cardiac Bioprostheses. For information on this meeting, contact Beverly Farrier, ProMedica International, 620 Newport Center Dr, Suite 575, Newport Beach, CA 92660; or telephone (714) 640-5870.

Ninth World Symposium on Cardiac Pacing and Electrophysiology, Washington, DC—May 28–31, 1991

This meeting will incorporate the Twelfth Annual Scientific Session of the North American Society of Pacing and Electrophysiology. For information on this meeting, contact IXth World Symposium Secretariat, c/o NASPE, 377J Elliot St, Newton Upper Falls, MA 02164; or telephone (617) 237-1866 (facsimile: (617) 431-1991).

The International Society for Cardiovascular Surgery/The Society for Vascular Surgery, Boston, Massachusetts—June 3–5, 1991

For information on this meeting, contact the Association offices, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Cardiac Arrhythmia Surgery International Seminar II, Geneva, Switzerland—June 12–14, 1991

For information on this meeting, contact Beverly Farrier, ProMedica International, 620 Newport Center Dr, Suite 575, Newport Beach, CA 92660; or telephone (714) 640-5870 (facsimile: (714) 759-6911).

Seventeenth Annual Meeting of The Western Thoracic Surgical Association, Seattle, Washington—June 26–29, 1991

For information on this meeting, contact The Western Thoracic Surgical Association, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Twentieth World Congress of The International Society for Cardiovascular Surgery, Amsterdam, the Netherlands—September 1–6, 1991

For information on this meeting, contact the Registrar: XX World Congress—ISCVS, International Travel Service, Inc, 104 Wilmot Rd, Suite 300, PO Box 825, Deerfield, IL 60015; or telephone (708) 940-2100 (facsimile: (708) 940-2386); or contact the Congress Secretariat, International Society for Cardiovascular Surgery, 13 Elm St, Manchester, MA 01944; or telephone (508) 526-8330 (facsimile: (508) 526-4018).

Heart Valve Replacement—Past Performance: Future Prospects, Sheffield, United Kingdom—September 19–21, 1991.

For information on this meeting, contact Jenny Pupius, Symposium Secretariat, Department of Medical Physics and Clinical Engineering, Royal Hallamshire Hospital, Glossop Rd, Sheffield S10 2JF, United Kingdom; or telephone (0742) 555250 (facsimile: (0742) 729981).

CLASSIFIED ADS

Classified ads for positions wanted or available must relate only to the thoracic or cardiovascular surgical specialties. Display space (minimum, ¼ page) is also available through Arthur Carlucci, Elsevier Science Publishing, 655 Avenue of the Americas, New York, NY 10010 (telephone: (212) 633-3806; facsimile: (212) 633-3880). All ads are subject to the approval of the editor, and material deemed inappropriate for publication in *The Annals* will be rejected. If anonymity is desired, please indicate this at the time of submission and a code number will be assigned. All responses received will remain confidential in the publisher's office and will be conveyed to the advertiser shortly after receipt. The charge for all classified advertising is **$1.25 per word per insertion, minimum 20 words.** Abbreviations, dates, initials, post office box numbers, telephone numbers, years, and zip codes are considered one word each. **There is an additional fee of $17.00 per insertion for box number ads.** The copy deadline is 7 weeks prior to publication, eg, for the March issue, copy should be received by the 1st of January. Ad orders should be for a maximum of 6 months; ads may be renewed for up to 6 months at a time. Ad copy should be typed double-spaced and mailed in duplicate to: *The Annals of Thoracic Surgery*, Desk Editorial, Classified Ads, Elsevier Science Publishing Co, Inc, 655 Avenue of the Americas, New York, NY 10010 (facsimile: (212) 633-3853). Make *nonrefundable* check payable to Elsevier Science Publishing Co, Inc.

Please note that *The Annals* does not function as a placement service and cannot provide information on advertised positions. When answering advertisements, please write individually to each advertiser you wish to contact, using the address given in the ad. To respond to an anonymous ad, write to Box Number, *The Annals of Thoracic Surgery*, Elsevier Science Publishing, 655 Avenue of the Americas, New York, NY 10010.

SITUATIONS AVAILABLE

Cardiovascular and thoracic surgeon, board eligible or certified, wanted to join another surgeon in private practice in the Southeast. Please respond with curriculum vitae.

Please respond to Box 252. 252G/L

Cardiothoracic surgeon needed for established program in scenic Montana. Unique opportunity for congenial private practice lifestyle combined with high-tech hospital environment including dedicated cardiac OR, cardiac anesthesia, and cardiac intensive care unit. Solo practice or association leading to partnership. Could be experienced surgeon or recent graduate from quality program.

Please respond to Box 354. 354G/L

Thoracic surgeon wanted: A busy thoracic practice 20 miles south of Boston seeks to add a third surgeon by July 1991. Candidate must be board certified or eligible. This active practice operates from a multihospital referral base. The community is an ideal area for family living, with excellent schools and nearby Boston and Cape Cod cultural and recreational facilities. Competitive salary, malpractice insurance, and excellent benefit package. Please respond with CV.

Please respond to Box 356. 356G/L

Cardiac surgeon, board certified or board eligible, for very busy private practice group doing primarily adult cardiac and vascular surgery. Seeking an Associate to join well-established practice in northeast Ohio metropolitan area. Please respond with curriculum vitae and references.

Please respond to Box 370. 370H/A

Cardiothoracic surgeon, ABTS certified/eligible, with primary interest in cardiac surgery, wanted immediately to replace member of established surgical group practicing in Allentown, PA. Competitive salary/fringe benefits. Position leads to partnership for successful candidate.

Please send curriculum vitae, clinical experience, and references to Joan M. Persa, Administrator, 1275 South Cedarcrest Blvd, Allentown, PA 18103; (215) 821-8790. 393I/B

Cardiovascular and thoracic surgeon: BE/BC wanted to join well-established private group practice in Philadelphia area. Seek recent or upcoming graduate. Please send curriculum vitae.

Please respond to Box 394. 394I/B

Thoracic, vascular, and general surgeon to join a busy, all boarded, private practice on eastern Long Island. Should be board certified or eligible. Excellent salary and benefits, leading to a full partnership. Send curriculum vitae.

Please respond to Box 395. 395I/L

Cardiovascular surgeon to become second surgeon in new (two year old) adult cardiac program on west coast of Florida.

Submit CV to Manatee Cardiac Surgeons, PA, 201 4th Ave East #2, Bradenton, FL 34208. 396I/B

Cardiovascular surgeon, board certified/eligible, wanted to join two surgeons in an established cardiovascular program in the southeastern USA. Must have experience in adult cardiac, thoracic, and vascular surgical procedures. Salary/full partnership negotiable. Please send resume.

Please respond to Box 398. 398I/B

Cardiovascular and thoracic surgeon: Excellent opportunity for a board certified/eligible cardiovascular and thoracic surgeon to join in a new, growing department in a metropolitan New York community on Long Island. Academic/private practice setting with excellent compensation. 533-bed community teaching hospital. Experience in electrophysiologic surgery, artificial devices, and/or transplantation helpful. Please respond with curriculum vitae.

Please respond to Box 403. 403I/L

Thoracic/vascular surgeon needed to join prominent, active 3-man group in desirable suburban community in greater NY metropolitan area. Excellent opportunity for well-trained, highly motivated individual. Recent graduate preferred. Possible academic affiliation. Please respond with CV.

Please respond to Box 404. 404J/C

Adult cardiac, peripheral vascular, and thoracic surgery practice in Midwest with rapidly growing referral base desires well-qualified surgeon for association leading to partnership. Practice includes busy transplant program. Academic affiliation available. Send CV and three references.

Please respond to Box 407. 407J/L

Thoracic and cardiovascular surgeon, board certified or eligible, wanted for an established practice in the Midwest with experience in adult cardiac, thoracic, and peripheral vascular surgery. Experience or training in heart/lung transplantation preferred. Initial contact should include curriculum vitae, clinical experience, and names of references.

Please respond to Box 408. 408J/B

Assistant surgeon—Busy adult and pediatric cardiac and vascular surgery group in suburban Washington, DC, is seeking a non-board certified surgeon, preferably with cardiovascular experience, to assist in the operating room. Excellent salary and fringe benefits.

Please respond with CV to Virginia Heart Surgery Associates, 3301 Woodburn Rd, Suite 301, Annandale, VA 22003. 413J/L

Cardiac surgeon—board certified/eligible to be third member of established practice in suburb of Washington, DC, expanding to second hospital. Excellent compensation and benefits. Great

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opportunity for recent graduate wishing to locate in area of nation's capital. Practice focus is primarily in adult cardiac surgery with additional work in thoracic and peripheral vascular surgery available.

Please send curriculum vitae to PO Box 34945, Bethesda, MD 20817. 415J/C

Cardiovascular surgeon (BC/BE) to join 2 recently trained, board certified associates in a well-established adult cardiac surgery program in a multispecialty clinic affiliated with a stable referral system of 24 satellite clinics. Active program includes arrhythmia surgery. Midwest location is 30 minutes from the Minnesota lakes district.

Contact Dakota Medical Center, 1702 South University Dr, Fargo, ND 58103; Dr John Hicks, Director, Recruiting Division, (701) 280-3338. 416J/C

Cardiothoracic/vascular surgeon: board certified in general and thoracic surgery. Minimum of 2 years of practice experience. Very active private 5-surgeon multispecialty practice. Located in western Pennsylvania. Salary leading to full partnership. Excellent benefits. Detailed CV must accompany response. All replies treated confidentially.

Please respond to Box 419. 419J/C

Cardiothoracic surgeon—to join a 2-man private practice in cardiac, thoracic, and vascular surgery in the Great Lakes region. Must be board certified or eligible. Recent graduates considered. Send curriculum vitae.

Please respond to Box 421. 421J/C

Cardiothoracic surgeon needed to join established 2-man group in practice of adult cardiac, pulmonary, and vascular surgery. Must be board certified or eligible and graduate from a US medical school.

Please respond to R. W. Ruess, MD, 1834 Broadway, Cape Girardeau, MO 63701. 422J/C

Cardiovascular-thoracic surgeon—BE/BC to join established and growing 2-surgeon group in northeastern US. Practice includes high volume cardiac and general thoracic surgery. Recent trainee preferred. Submit CV.

Please reply to Box 423. 423J/C

Cardiothoracic surgeon wanted for busy, expanding private practice in Connecticut. Practice includes cardiac and thoracic surgery. Surgeon must be board eligible or certified. Excellent opportunity for suitable applicant.

Please respond to Box 424. 424K/A

Pacific Northwest opportunity for cardiothoracic surgeon with 2 to 4 years of experience. Growing multispecialty group practice in Portland, Oregon, seeks dynamic surgeon to join 3 other surgeons in our practice. General thoracic and vascular experience necessary.

Please send curriculum vitae to Dr James W. Asaph, MD, 507 NE 47th Ave, Portland, OR 97213-2282. 426K/D

Cardiothoracic surgeon, recent graduate, experienced in cardiac arrhythmia surgery, to join well-established cardiac surgical group in central Florida. Send CV.

Please respond to Box 427. 427K/A

Cardiac surgeon wanted to join established private practice in upstate New York, primarily adult cardiac. Board certified or eligible required. New or recent graduate preferred. Please send CV and references.

Please respond to M. A. Marvasti, MD, 101 Union Ave, #813, Syracuse, NY 13203. 429K/D

Wanted: BE/BC general surgeon with interest and experience in thoracic surgery who enjoys general surgery, or non-cardiac thoracic surgery, to assume practice of retiring double-boarded thoracic/general surgeon. 34-member multispecialty group in dynamic area hub city of 35,000, with six satellite offices in strategic locations and referral area of 300,000 plus. Superb hospital facilities, including CT, MRI, cardiac surgery, neurosurgery, etc. Directed towards those concerned about quality family lifestyle and professional concerns.

Contact Recruitment Coordinator, Park Clinic, 890 North Eisenhower Ave, Mason City, IA 50401; or dial 800-798-4321 for more information. 431K/D

Wanted: BC/BE cardiac surgeon to join an active cardiac surgical group in the Midwest. All aspects of adult and pediatric cardiac surgery performed along with an active artificial heart and transplant program.

Please respond to Box 434. 434K/L

Cardiovascular/vascular surgeon wanted to join busy established adult practice in Ohio. Recent graduate preferred. Please submit curriculum vitae. Association will lead to partnership.

Please respond to Box 436. 436K/C

BC/BE adult cardiothoracic surgeon wanted to join active established department in large multispecialty group practice located in metropolitan Midwest community. Medical school affiliation available. Respond with curriculum vitae.

Please respond to Box 437. 437K/D

Minneapolis cardiothoracic/vascular group seeks to add fifth surgeon, BC/BE.

Respond to BLH Physician Search, 2231 Lee Ave N, Minneapolis, MN 55422; (612) 588-1116 or -0333. 438L/E

Board certified/eligible cardiothoracic surgeon for busy 1-physician practice in Atlanta. Transplant experience. Future partnership.

Send resume to Georgia Cardiac Surgical Association, PO Box 491203, College Park, GA 30349. 439L/E

The Department of Surgery at the University of California, Davis, seeks a full-time academic pediatric cardiac surgeon. The level of appointment will be commensurate with qualifications. Applicants must be board certified or board eligible in thoracic surgery and have specialized training in pediatric cardiac surgery.

A letter outlining research and teaching background, a curriculum vitae, and bibliography, along with the names and addresses of three references should be forwarded to Herbert Berkoff, MD, Chief of the Section of Cardiac Surgery, Department of Surgery, University of California, Davis, 4301 X St, Sacramento, CA 95817. Applications will not be accepted after April 30, 1991. The University of California is an affirmative action/equal opportunity employer. 440L

Thoracic surgeon, BE/BC, wanted to join 4-man group on Gulf Coast of Florida, specializing in general, vascular, and noncardiac thoracic surgery. Recent graduate preferred. Please send CV and references.

Please respond to Box 441. 441L/B

Cardiothoracic surgeon wanted for July 1991: expanding private practice in Philadelphia. Adult cardiac and thoracic surgery. Academic appointment. Eventual partnership.

Send CV and references to PO Box 1295, Philadelphia, PA 19105. 442L/B

Vacancy exists for a BE/BC thoracic and cardiovascular surgeon. Candidate must have proven clinical and laboratory abilities and scular surgery fellowship. Training required in major complex ascular procedures.

lease forward curriculum vitae to VAMC, Attention: Carolyn G. Gray, 2002 Holcombe Blvd, Houston, TX 77030; or call (713) 795-7463. EOE. 446L

Cardiothoracic/vascular surgeon: Recently trained, board eligible or board certified surgeon wanted to join expanding practice in central California. Excellent community, competitive salary leading to partnership. Please respond with CV and references.

Please respond to Box 447. 447L

Cardiovascular and thoracic surgeon, BC/BE: Exciting academic position available for the initiation of a cardiac transplant program, participation in ongoing clinical and basic science research, and a busy clinical cardiac and thoracic surgical practice. An extremely competitive salary and benefit package is available.

Candidates should forward a letter of application and CV to Cardiovascular and Thoracic Surgery, University of Kentucky Medical Center, 800 Rose St, Lexington, KY 40536-0084. 448L/A

Cardiovascular/thoracic surgeon, BC/BE, prefer recent graduate to join 2-man group for private practice, predominantly in cardiac surgery in Orange County, southern California. Excellent salary. Send CV with experience and recommendations.

Please respond to Box 449. 449L

Wanted—thoracic surgeon, BE/BC, to join a board certified surgeon for noncardiac thoracic surgery and peripheral vascular surgery in a growing medical center in upstate New York.

Please respond to Box 451. 451L/B

Chief, Cardiothoracic Surgery: Excellent opportunity to lead cardiothoracic surgery department for 400-bed community teaching hospital located in western Pennsylvania. This dynamic facility performs 900 open heart procedures and 5,600 catheterizations per year. Qualified candidates should possess academic credentials and have interest in developing clinical research.

Please contact Jeanne Dowrick, Fulton, Longshore and Associates, Inc, 527 Plymouth Rd, Plymouth Meeting, PA 19462; telephone (800) 346-8397 or (215) 834-6780. 453L/B

Thoracic and vascular surgeon needed—Southeast: Looking for a partner to join an established surgeon to practice noncardiac thoracic and vascular surgery in mid-sized city. Must be board certified or eligible. No buy-in needed, guaranteed income available. Excellent community to raise a family with the cultural advantages of a large city but not the hassles. Will consider others, but recent graduate preferred. Send CV, and include description of interests, and career goals.

Please respond to Box 454. 454L/C

Cardiovascular and thoracic surgeon: Position available for a board certified or eligible cardiovascular and thoracic surgeon to join a 2-man private practice involved with adult cardiac, thoracic, and peripheral vascular surgery in a desirable eastern metropolitan area. Association features competitive salary, excellent fringe benefits, and a goal of early partnership. Please send curriculum vitae.

Please respond to Box 455. 455L/B

Surgeon with interest/experience in hemodialysis access to manage 400–450 cases per year as part of a large, established, mid-Atlantic cardiovascular and thoracic surgical practice. ABS required; salary, incentives, and call schedule negotiable. Send CV.

Please respond to Box 456. 456L/B

Cardiothoracic surgeon interested in joining an established practice in the Southwest. Must be experienced in adult cardiac, pulmonary, and vascular surgery. Must be board certified/board eligible. Please respond with curriculum vitae.

Please respond to Box 457. 457L/A

Thoracic and cardiovascular surgeon wanted to join a busy practice. Prefer recent graduate. Must be board certified or eligible. Located in Midwest. Please respond with curriculum vitae.

Please respond to Box 458. 458L/B

Cardiovascular and thoracic surgeon, BE/BC, to join another physician in a rapidly expanding private practice located in the Southeast. Please send curriculum vitae and references.

Please respond to Box 459. 459L/E

University of Massachusetts Medical Center is recruiting candidates for its newly opened Cardiothoracic Surgical Residency Program for 1991, 1992, and 1993. The applications for the 1993 position will be processed through the Match Program for Cardiothoracic Residencies.

Request applications from and submit curriculum vitae and three letters of recommendation to Thomas J. Vander Salm, MD, Chief, Division of Cardiothoracic Surgery, University of Massachusetts Medical Center, 55 Lake Ave North, Worcester, MA 01655; telephone: (508) 856-2216. 462L/E

Cardiovascular surgeon to join a young diplomate, ABTS, in a practice that has grown to some 600 hospital surgeries per year. Location is in a city in the mid-South. The only open heart program in 15 counties. 325-bed regional medical center. Two catherization labs. Practice consists of cardiac, thoracic, and vascular surgery. Full financial support including productivity incentive. All benefits, malpractice and office expenses provided.

Please respond to Box 463. 463L

Cardiac surgeon, board certified or eligible, needed for a growing cardiac surgery program in the Southeast. Excellent opportunity for someone primarily interested in adult cardiac surgery.

Please respond to Box 464. 464L/A

SITUATIONS WANTED

Cardiovascular and thoracic surgeon, university trained, 39, hard working, proficient in valve repairs, IMA grafting, redo surgery, experienced, wishes to relocate. Also available, cardiovascular anesthetist or intensivist to make self-sufficient team to start new practice. All inquiries handled with strictist confidence.

Please respond to Box 428. 428K/A

Cardiovascular and thoracic surgeon, 40, ABTS certified, university trained, experienced in establishing new program, seeks solo or group practice opportunities.

Please respond to Box 435. 435K/L

Cardiovascular/thoracic surgeon, 52, university trained. Busy private practice in same location for 20 years. Established cardiac program, President of Medical Staff, ABS/ABTS, vascular board, critical care board. Seeks private practice opportunity due to local hospital merger.

Please respond to Box 450. 450L/A

FELLOWSHIPS

Fellowship in cardiovascular surgery for 1 year available January 1 or July 1 with group doing approximately 1,000 cardiac procedures per year and large volume of vascular procedures. ABS eligible or certified, California medical license required or readily obtainable. Ideal for a person between general surgery residency and thoracic surgery residency.

LIBRARY

Please respond with curriculum vitae to Bradley J. Harlan, MD, 5301 F Street, Suite 312, Sacramento, CA 95819. 364G/L

Fellowship in pediatric cardiovascular surgery available July 1991. Experience includes surgery for hypoplastic left heart syndrome and arterial switch. A large, well-organized collection of heart specimens available for review. Illinois license or equivalent required. Salary and benefits in the high $40s.

Please respond to Division of Pediatric Cardiac Surgery, The Heart Institute for Children, Christ Hospital and Medical Center, 4440 W 95th St, Oak Lawn, IL 60453. 443L/C

Fellowship in vascular surgery—The Deborah Heart & Lung Center is currently seeking qualified applicants for the position of a Vascular Fellow beginning January 1, 1991.

Please contact Douglas H. Joyce, DO, Director, Surgical Residency/Fellowship Programs, 200 Trenton Rd, Browns Mills, NJ 08015; (609) 893-6611, ext. 359. 444L

Surgical residents/fellows—Clinical residency/fellowship positions available in adult and pediatric cardiothoracic surgery. Large volume of cases. New Jersey license required. Excellent salary and benefits.

Please contact Lynn B. McGrath, MD, Chairman, Department of Surgery, Deborah Heart & Lung Center, 200 Trenton Rd, Browns Mills, NJ 08015; (609) 893-6611, ext. 359. 445L

Fellowship in cardiovascular surgery: Fellowships for 1 to 2 years with busy service performing many complex cardiac procedures. 1989 cases—1,300. Opportunity to rotate in pediatric service. Foreign graduates must have ECFMG/FMGMS or be eligible for H-1 visa. US and Canadian graduates must be eligible for Oregon licensure.

Please send complete CV to Dr Albert Starr, 9155 SW Barnes Rd, Suite 240, Portland, OR 97225. 452L/B

Cardiopulmonary surgery fellow: July 1991 opening of a clinical position for 1 year in a busy university service that performs cardiac and thoracic surgery, pediatric heart surgery, and cardiac transplantation. The fellow will be given responsibilities in perioperative care and as assistant and operating surgeon based on prior experience. Ideal position for a senior surgical resident who would like to attain additional experience in cardiopulmonary surgery or a resident waiting to enter a thoracic surgery residency.

Please respond with curriculum vitae to Adnan Cobanoglu, MD, Oregon Health Sciences University, 3181 SW Sam Jackson Park Rd, Portland, OR 97201-3098. 460L/A

A 2-year Cardiovascular Research Fellowship position is available 7/1/91 in the Division of Cardiothoracic Surgery at the University of Massachusetts Medical Center. This is an opportunity for a closely supervised and highly productive cardiac research experience. Requirements: (1) A minimum of 2 years of surgical training; (2) a commitment from an institution to complete the applicant's training in general surgery; (3) intentions to pursue an academic career in cardiothoracic surgery.

Interested candidates should contact Marc S. Visner, MD, Division of Cardiothoracic Surgery, University of Massachusetts Medical Center, 55 Lake Ave North, Worcester, MA 01655 telephone: (508) 856-2975. 461L/B

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West Virginia University (WVU) Health Sciences Center, Department of Anesthesiology, is recruiting for a pediatric cardiac anesthesiologist. Our Department currently has 15 faculty members and sponsors an approved residency training program, which has 24 residents.

Requirements for entry as Assistant Professor include completion of CA III or fellowship year, with Board Certification preferred. An outstanding teaching opportunity is provided as resident and medical student education represent the primary mission of the Department. Related responsibilities include clinical anesthesia with a rare opportunity to provide anesthesia for complex congenital heart surgery in neonates, like hypoplastic left heart syndrome and arterial switch procedures. The Pediatric Cardiac Surgery Service performs 180-200 cases per year. Seven pediatric cardiologists cover the entire state through outreach clinics. Opportunities are available for collaborative research projects with the Section of Pediatric Cardiology and the Pediatric Cardiac Surgery Service.

This position requires selection of the tenure track, with its concerted research commitment. Time and start-up monies will both be available to support this research.

WVU is in Morgantown, West Virginia, approximately 75 miles south of Pittsburgh on major interstate highways. The area, providing abundant recreation for all seasons, is situated in the scenic Alleghany Highlands region.

Clinical services are provided at the Ruby Memorial Hospital, a tertiary care center which opened in July, 1988. This $87 million, 376-bed facility features 12 operating rooms, all with state-of-the-art equipment.

As evidence of tremendous institutional growth is the new $25 million Physician Office Center, which houses the largest multi-specialty group practice in the State; the new Cancer Center, which is the State's only NCI-designated cancer research center; Chestnut Ridge Hospital, a 70-bed psychiatric hospital for comprehensive inpatient and outpatient care; and a 60-bed rehabilitation hospital, now under construction.

WVU is an Equal Opportunity/Affirmative Action Institution.

For additional information, contact:

Edward Stullken, M.D., Chairman
Department of Anesthesiology
WVU Health Sciences Center
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Telephone: (304) 293-5411

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The University of Massachusetts Medical Center opened its Cardiothoracic Surgical Residency in July, 1990.

We are currently recruiting applicants for positions open in 1991, 1992, and 1993. The applications for the 1993 position will be processed through the Match Program for Cardiothoracic Surgical Residencies.

Interested candidates should request an application form, forward a curriculum vitae, and arrange for three letters of recommendation to be sent to:

Thomas J. Vander Salm, M.D.
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